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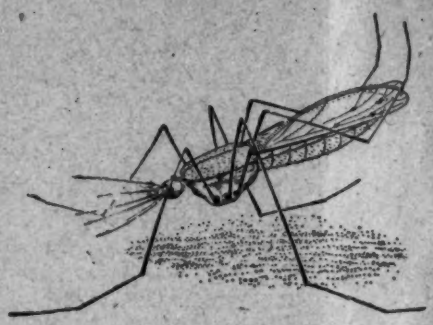

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BRITISH MEDICAL JOURNAL

LONDON SATURDAY JULY 23 1949

FIBRO-FATTY TISSUE AND ITS RELATION TO CERTAIN "RHEUMATIC" SYNDROMES*

BY

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If the observations I here put forward are correct, we are now in a position to turn over a fresh page so far as our conception of certain of the so-called rheumatic syndromes is concerned. I am not able to do much more than write the chapter heading for this new page; it seems to me, however, that this new chapter will be worth writing when adequate material is forthcoming.

It is generally agreed that much of the pathology described for the chronic rheumatic diseases is speculative rather than factual. Biopsy does not seem to confirm the current impression that muscle or fibrous tissues are generally the seat of cellular change in non-articular rheumatism, nor indeed that inflammation is the underlying pathology.

So far as aetiology is concerned, I suggest that bacteriology has had its day, and that the day of endocrinology and perhaps genetics is dawning. The therapeutic witch-hunt for focal sepsis is slackening—we need an alternative line of thought.

Pathologically, the contribution I wish to make is simple but, I believe, fairly far-reaching in this field. There is a third tissue, also of mesodermal origin—namely, fat—which has seldom been considered in this connexion, but which seems to be quite commonly subject to pathological variations which may cause symptoms that are labelled "rheumatic." Fatty tissue is widely distributed and constitutes nearly 20% of the body weight. It appears to be functional as well as protective, and, this being so, it would be strange if its functions were not sometimes disordered. One of these functions seems to be concerned with water storage (the most striking example of this is perhaps seen in the behaviour of the camel's hump). It has been established of recent years by Bishop and others that human fat may retain water to a pathological degree in certain disorders. It would seem to be some derangement of this normal function of water storage which underlies the syndromes discussed here.

Fibrositis

Fibrositis as a clinical entity has for many years past been accepted in this country, although its pathological foundations are, as has been mentioned, slender, resting almost entirely upon the theories of Gowers (1904), the equally unconfirmed histological studies of Stockman (1920), and the suggestion of Elliott (1944) regarding muscle spasm as a cause. Stockman defined fibrositis as "a condition of chronic inflammation of the white fibrous tissue of the fascial aponeurosis, sheaths of muscles and nerves, ligaments, tendons, periosteum, and subcutaneous tissues, occurring in all parts of the body and giving rise to pain, aching, stiffness, and other symptoms, the result of preceding general infections or local inflammations or injuries." It is now generally defined less comprehensively as an idiopathic disorder of the soft tissues chiefly characterized by pain and tenderness, which may be local, widespread, or referred, and is often associated with muscular spasm.

Nature of the Pain in Fibrositis

It was only comparatively recently that Lewis and Kellgren and others pointed out that the pain in fibrositis generally has its origin in certain focal points from which the more general subjective pain complained of by the sufferer is referred according to a segmental plan. This referred pain may seem to be situated at a considerable distance from its real origin. A good example is to be seen in those cases of sciatica not due to a disk mechanism, in which the focal point of pain—the "trigger-point"—may be found in the region of the iliac crest or even in the lumbar region. These trigger-points, which when palpable are generally termed "fibrositic nodules," are definite objective clinical entities in so far that when they are found by the examining finger the patient winces involuntarily. They are not merely tender spots, which are also commonly found, but they are specific points at which real pain is produced, and from which the more general pain can be referred by quite light pressure. The existence of these trigger-points is now well recognized in view of the therapeutic success which often attends their injection with local analgesics in saline. Palpable nodules which are not tender are not infrequently found, but do not appear to be significant from a clinical point of view.

Classification of Fibrositis

Himsworth, in his 1949 Oliver–Sharpey Lectures, has emphasized the liberation of medical thought which has resulted from the substitution of syndromes for "disease entities" as units of illness. The syndrome has its philosophical basis in a chain of physiological processes, interference with which at any point produces the same impairment of bodily function. He points out that the same syndrome may thus arise from different causes. This conception inspires a much more catholic concept of aetiology, which is no doubt fully applicable to the field of "rheumatism" in all its forms. It must not therefore be assumed that the conclusions put forward here are thought to explain more than a certain proportion of cases. It will also be apparent that an aetiological classification will be unlikely of attainment in this field, where causes are probably multiple.

It would seem that the only objective sign of the disorder lies in these localized trigger-points, which are, moreover, sometimes palpable in the form of nodules; it therefore seems reasonable to attempt to classify idiopathic fibrositis according to the anatomical nature of the nodule where this is known or strongly suspected. Very little biopsy or

*Hunterian Oration delivered at the Royal College of Surgeons of England on March 25.

other work of this nature has been done as yet, and the accompanying table based upon this objective must be regarded as being very provisional. It seems possible, however, that some form of classification such as that which follows might ultimately prove useful.

Classification of "Fibrositis"

Nature of Pain	Site	Clinical Finding	Morbid Anatomy	Cause
1. Focal ..	Tendon or aponeurosis	Nodule, small and hard	Fibrous	Trauma, rheumatic fever
2. Focal ..	Lumbar or gluteal	Larger nodule	Fibro-fatty	Oedematous changes
3. Focal ..	On muscle belly	Smallish elongated "nodule"	Local spasm of muscle fibres	Irritative focus in neural segment
4. Diffuse ..	Whole muscle group	Muscle tenderness	Unknown	Toxaemia
5. Diffuse ..	Many muscle groups	Spasm	Chronic muscular fatigue	Psychoneurosis
6. Diffuse .. (sometimes local)	Subcutaneous tissues	Panniculitis, tender fat deposits	Abnormal oedematous deep fat	Endocrine

Aetiology of Trigger-points and Nodules

It was observed (Copeman, 1943) that the pain in the back which accompanies most pyrexial illnesses is of the same nature and pattern as in fibrositis. It was shown, moreover, that, although the pain disappears with the cessation of pyrexia, the tender trigger-point will persist in a proportion of cases often for very long periods, unknown to the patient. These trigger-points can be reactivated by a recurrence of pyrexia, even if this is due to a different cause. It is therefore suggested that the tissues in which trigger-points and nodules subsequently occur in the course of fibrositis may sometimes have been "sensitized" earlier in life in the course of an attack of influenza or one of the exanthemata. The nature of the lesion may be a recurrent oedema painfully distending certain fat lobules in the deep subcutaneous tissues. It has also seemed certain that trauma, and probably also the direct action of cold, can be causative of similar lesions or at least precipitate their development in this way. No evidence of the direct causative effect of focal infection has been established.

Fibrositis Due to Oedematous Changes in the Fibro-fatty Tissues in Certain Sites

Method of Investigation

The frequency of the occurrence of "fibrositic" pain of the lower back in otherwise healthy young soldiers first stimulated our interest in this matter. As a first step exact measurements were taken of the site of these trigger-points or nodules in a large number of sufferers and a pain chart (Fig. 1) was plotted. The back of every patient who died in hospital was systematically dissected and special reference was made to these areas. As the result of this, and of many subsequent clinical observations and biopsies, certain conclusions regarding "fibrositis" and similar conditions in this and other regions of the body have been arrived at, many of which are embodied in this paper. The pain chart which evolved as the result of plotting the site of the trigger-points or painful nodules in our series of cases of lumbar fibrositis, as already mentioned, was found to outline the erector spinae muscles, the crest of the iliac bones, and the sacro-iliac joints. It was later found that this chart also corresponded with areas in which residual fat occurred, even in patients dead with the grossest forms of cachexia.

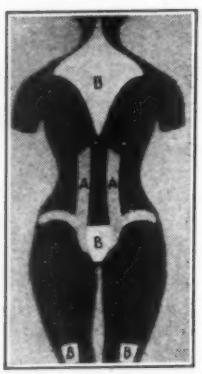

FIG. 1.—Usual sites of fibro-fatty lesions. A, herniation. B, tension pain.

As no evidence of pathological change could be found in fibrous tissue or muscle in biopsy material, we decided to examine the deeper layers of fat which constituted the remaining tissue in the neighbourhood of the trigger-point —notwithstanding the usual assumption that no pathology of interest normally affects this type of tissue. We were lucky in our first case in that we noticed a curious herniation of a large lobule of distended fat through a defect in the deep fascia. In a later biopsy of an easily felt and tender nodule from which widespread pain was being referred we discovered by cautious dissection that the nodule in this case was an oedematous-looking node of fatty tissue lying among superficial fat of the upper buttock, but with a pedicle which could be traced down to the layer of fat lying beneath the deep fascia.

Fatty Structures as a Site for Painful Lesions

In our original paper (Copeman and Ackerman, 1944) we described the areas of fat in which painful "fibrositic" lesions occurred as being in the main "residual," in the sense that lobulated fat remains in these areas even when it disappears from other areas as the result of extreme cachexia. The colour of this residual fat in such cases was often darker or pinker than ordinary fat. In two cases we were able after death to trace the pedicle of a superficial fat hernia down to the paranephric fat mass. These observations suggested to us that the areas of fat liable to be affected, at any rate in the lumbar region, might bear some relation developmentally to the primitive renal fat gland round which, from a comparatively early period of embryonic development, fat congregates in closely lobulated clumps, which in rats (according to Hammar) are also of a darker colour than normal. The well-marked area of residual fat which lines the rim of the ilium may not be covered by this hypothesis, although another primitive fat-organ is described in the outer groin in some textbooks. The largest of these fat-organs is said to be situated between the scapulae—a position which seems to correspond with that of our "dorsal fat-pad."

Normal Anatomical Disposition of Fat

Not much information is to be found on this subject in most anatomical textbooks. Subcutaneous fat exists very generally throughout the body, although its distribution is not uniform. In certain situations it collects more abundantly. Here it forms a considerable layer beneath the reticular layer of the corium, where it is laid down in the subcutaneous areolar tissue—panniculus adiposus. This areolar tissue forms a thin, indistensible fibrous capsule round the large lobules, where the fat is lobulated. The lymphatics serving fat accompany the blood vessels in very close relation as they enter the lobule. There are numerous sensory nerves to the blood vessels, and possibly to other structures in the lobules, and sympathetic-nerve fibres which run to the fat cells control the function of storage and yielding. Where the skin is thicker and less movable the reticular layer is fixed to the deep fascia by numerous stout fibrous bands (retinaculum cutis), the space between being filled with firm fat-clusters. This formation normally occurs mostly on the posterior aspects of the trunk and the upper and outer aspects of the limbs.

Pathological Herniation of Fat Lobules

From the brief account which has so far been given it will appear that what pass as fibrositic trigger-points can often be shown to be related topographically in the lumbo-sacral region with the basic fat pattern. Where these tender points were surgically explored, herniation of distended fat lobules through deficiencies in their investing fibrous covering was often found. Removal or disruption of these

herniae in such cases provided lasting relief from pain. Histological examination of the material removed has shown in practically every case nothing but normal fat tissue distended with œdematous fluid—suggesting that the cause of this oedema is not inflammatory.

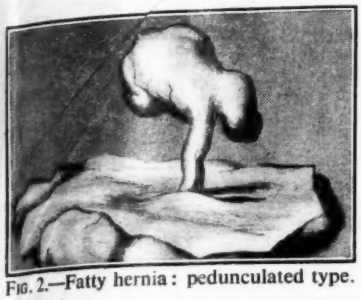

FIG. 2.—Fatty hernia: pedunculated type.

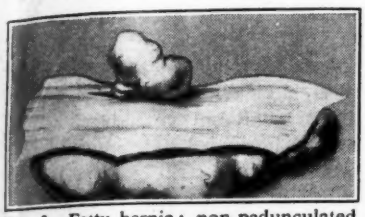

FIG. 3.—Fatty hernia: non-pedunculated type.

FIG. 4.—Fatty hernia: foraminal type.

In the lumbo-sacral region the fat herniae seen so far have been of three types: (*a*) pedunculated (Fig. 2), (*b*) non-pedunculated (Fig. 3), and (*c*) foraminal (Copeman and Ackerman, 1944) (Fig. 4). The first two are self-explanatory, the herniation occurring from the deeper layer of fat through a flaw in the fascial covering or layer into a more superficial layer. The foraminal herniae occur along the foramina which exist in the deep fascia of the sacro-spinalis muscles, along which pass the lateral branches of the posterior primary divisions of the first, second, and third lumbar nerves together with a small artery and vein.

Twenty-two selected case histories from our series have been published (Copeman and Ackerman 1944, 1947) in which removal of a fat hernia of one of these types resulted in a permanent cure of the pain, and many subsequent unpublished cases have been investigated with similar findings and results. Herz (1945), working in Cleveland, has confirmed this work and has published two further, larger series of successful cases.

Lesions Other than Herniation Occurring in Fat of Normal Distribution

Upper Dorsal Region

The skin is normally six to eight times thicker in this situation than in most parts of the body, and islands of fat may be found actually in the reticular layer of the corium of patients suffering from panniculitis. In the upper back the fibrous projections between the corium and the deep fascia normally form a series of honeycomb-like compartments which are filled with vascular fat. The axes of these "cells" are found to be roughly parallel with the tension (cleavage) lines of Langer. This structure is particularly noticeable in the upper back area, and we have called this diamond-shaped area the "dorsal fat-pad." It is one of the common sites of "fibrositic" pain, and its area corresponds fairly accurately with that of the trapezius muscle which lies beneath it.

When painful nodules are palpable, as is commonly the case in "fibrositis" of this region, they are seldom fibrous in nature. Biopsy will generally disclose the nodule to consist of the fatty contents of one or more of these compartments, swollen and under tension. Tension is shown by the fact that if the incision is made at right angles to the lines of tension in the skin the wound will gape and the lobule will be seen to present above its surface. It is also shown by the *peau d'orange* appearance if the skin and subcutaneous tissues of an affected area are compressed laterally. Negative evidence of this is afforded by the well-attested observation that palpable nodules of this type can be "rubbed away" by means of deep massage; a fact that could not be rationalized if they were formed of solid material such as fibrous tissue. Herniation of these lobules into adjoining compartments has been observed, but is uncommon owing to the density of their walls.

Periarticular Fat-pads

It does not seem to be generally appreciated amongst clinicians that localized collections of fat are to be found normally in association with certain joints, particularly the knees and ankles, and that these may become painful without the joint itself necessarily being affected. When this occurs an irresponsible diagnosis of arthritis is often made as the result of insufficient examination.

(*a*) *The Patellar Fat-pads.*—A normal intracapsular fat-pad is situated between the joint and the patella. This is well recognized by anatomists and surgeons. These infra-patellar pads will often become tender and painful without the joint itself being affected; this is particularly so around the time of the menopause. It is this lesion that commonly gives rise to the sensation of grating on movement, which is sometimes considered to imply that arthritis is present. A fold called the infrapatellar synovial fold extends posteriorly from the apex of each fat-pad to be attached to the femur inside the joint cavity. No doubt in certain cases, if this is also affected, intra-articular function will to some extent be deranged.

(*b*) *The "Popliteal Fat-pad."*—This has not, so far as I am aware, been described as a separate entity. None the less, it is present in more than 80% of normal people over

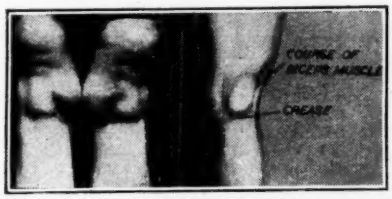

FIG. 5.—The popliteal fat-pads: normal structures which generally enlarge when painful. Often mistaken for bursae.

FIG. 6.—General obesity. A, Enlargement of popliteal fat-pads. B, Additional fat-pads at inner aspects of knees. (By courtesy of *Amer. J. med. Sci.*)

40 years of age. When it attracts notice by enlarging and becoming tender it is often wrongly described as a bursal enlargement (Figs. 5 and 6). It is sharply limited at its lower margin by the upper crease of the popliteal skin, and its outer border lies roughly parallel with the course of the semimembranosus muscle. Its upper border, when the pad is large, is delineated by another crease which develops in these circumstances. The inner border lies to the outer aspect of the midline of the popliteal space. This fat-pad lies between the superficial and deep layers of the fascia covering the popliteal space. When large it distends the skin, although the cause of pain and tenderness seems to lie rather in the distension of the fat itself. In these circumstances, however, full flexion of the knee-joint, by compressing the fat-pad, may give rise to pain—and so again to a mistaken diagnosis of arthritis.

The Sacral Fat-pad

This can be observed in about 10% of normal persons (Fig. 7). In structure it is very similar to the dorsal fat-pad. It is rather smaller than the sacrum, but is of the same shape and lies over it. It sometimes enlarges painfully as an isolated phenomenon, but is more often in association with a general condition of panniculitis, either with or without obesity.

FIG. 7.—The sacral fat-pads, present in about 10% of normal subjects.

Two other small fat-pads exist in many normal persons and in all who are obese. I can find no published description of these, however. The first is situated over the dorsal surface of the basal phalanx of the great toe. The so-called "male hairs" described by Wood Jones spring from its surface. It seems to be designed to buffer the strong action between the tendon of extensor hallucis longus and the skin. It is seldom of clinical significance, although its enlargement greatly contributes to the shiny swelling of the skin in this region in cases of acute and subacute gout. The other fat-pads which seem to be of some significance, in so far as they often become swollen and locally painful in obese persons, are situated at the ankle-joint. Medially there is one between the malleolus and the heel, roughly over the flexor retinaculum; whilst on the fibular side one lies over the stem of the inferior extensor retinaculum. A further fat-pad sometimes develops over the adductor tendons of the thigh at their pubic origin. It is rather doubtful, however, whether this can be considered to be of normal occurrence. It always develops in cases of obesity, and may be very painful.

Clinical Effect of Altering Volume and Distribution of Body Fluids

If the views which have been propounded are correct regarding the role of increased fluid tension in certain tissues causing pain, it should prove possible to relieve the pain by reversing this process. An experiment was accordingly planned (Copeman and Pugh, 1945) whereby 22 cases of fibrositis in which we believed that the pain might be of this type were selected. In these patients we induced the state of clinical dehydration; at which point we added electrolyte to the extracellular fluid to increase its osmotic pressure. Since the cell membranes are impermeable to sodium and chloride, intracellular dehydration would result.

The method of achieving this was to restrict the fluid intake of these patients for 36 hours to about 8 oz. (225 ml.). After this they were allowed neither food nor drink for 24 hours, and their fluid output was increased by the administration of ½ oz. (14 ml.) of sodium sulphate at hourly intervals for six doses. The process of dehydration could be followed by noting the daily loss of weight, which varied between 1 and 3.5 kg. This preliminary stage of dehydration was followed by an intravenous injection of 50 ml. of 30% sodium chloride (15 g.) into a vein. Immediately after this injection the patient was allowed to drink 4 oz. (114 ml.) of tea to mitigate thirst. Apart from this no fluid was permitted for four hours, after which no further limitation was imposed and the patient was encouraged to get up and about.

The clinical result of intracellular dehydration in this group was that of the 22 patients submitted 13 were rendered completely free of pain for variable periods. It was thought that this experiment confirmed other evidence about the nature of the mechanism of the pain suffered in the successful cases.

Relationship of Panniculitis with Fibrositis

According to Stockman pain of unknown origin occurring in mesodermal tissue, which he believed (as is indicated by his nomenclature) was always due to inflammation of the white fibrous tissues, is covered by the generic term fibrositis. Pain which has been described above as occurring from the distension in normally situated subcutaneous fat lobules can also therefore conveniently be classified under this heading. Where pain of this type occurs in abnormally deposited fat, however, it will be referred to as panniculitis. The condition was described by Stockman (1911) and also by Telling. It was defined by the former as being fibrosis of the deep subcutaneous tissues, or more specifically as "a chronic inflammation of the areolar tissue of the panniculus adiposus, including the small nerves and blood vessels." Although he recognized the presence of excessive fat locally he considered this to be the result of the inflammation and did not recognize that it had any association with the pain.

Panniculitis is a common condition which is insufficiently recognized as a cause of pain which is not infrequently labelled "psychogenic." Our investigations have shown no single instance histologically of an inflammatory reaction having occurred, and we believe that the pain in this condition originates as the result of oedema, and consequently distension, of lobules of the abnormal fat deposits. Panniculitis can best be considered in relation to those regions of the body in which it is most commonly encountered—namely, the upper dorsal region, the upper and outer aspects of the limbs, around certain joints, especially the knees, elbows, and ankles, and some other sites. Panniculitis is often associated with general obesity. Even in cases showing this association the sites of pain generally remain confined to the regions in which pain is situated in cases not so complicated. This continuing localization of the pain in cases of general obesity may be due to the less lobulated nature of the fat which is laid down elsewhere and also to the fact that in other sites, such as the abdominal wall, no fibrous fascia or capsule is present to limit the distension of the fat if it becomes oedematous. Herniation of fat lobules is common in association with obesity.

Although adiposis dolorosa would seem to differ from the preceding syndrome merely in degree, it does show more generalized areas of pain. The nodular feel of the fat deposits, the fluctuation in the sites of pain, and the histological findings, however, suggest that the underlying cause of the pain may be the same as in the preceding syndromes—namely, a shifting non-inflammatory oedema affecting the pathological fat deposits. In this connexion it is interesting to note in Dercum's (1892) original description that "occasionally very severe paroxysms of pain occurred, and these were coincident with temporary swelling and hardening of the fat masses."

Panniculitis of the Upper Dorsal Region

The "dorsal fat-pad," which has been described as the seat of local lesions in fibrositis, is more generally involved in cases of panniculitis. The tissues are sometimes excessively thickened and the subcutaneous tissues cannot be lifted between the finger and thumb. It is sometimes the only region involved in this process, in which case the condition tends to be concentrated round the base of the cervical spine—the "buffalo hump."

Panniculitis of the Upper and Outer Aspects of the Limbs

Tender nodules not infrequently form along the upper and outer aspects of the thighs, often in association with enlargement of the periarticular fat-pads of the knee, which are discussed below. Pain due to this cause is generally diagnosed as "fibrositis." A few biopsies and dissections have been made in order to study the method of formation of nodules in this site.

The usual situation of these localized collections of fat is in the panniculus adiposus, where certain portions of the areolar tissue seem to become more heavily charged with fat than do others. These portions appear to be more closely lobulated and independent of the average layer of fat at this level, although lying in it, and give the appearance of being small independent "mesenteries" each with its own blood and lymphatic supply. There is also a large potential space between the superficial and deep layers of the fascia which overlies the fascia lata of the thighs, and fat nodules sometimes form also in this space.

In cases in which these collections become painful in this situation it is found that they have become circumscribed by adhesions which seem to prevent lateral extension of the fat mass. The pain is presumably due in such cases to attempted distension of the unyielding fascial walls. The cause of these limiting adhesions is unknown; it may be traumatic, but since their occurrence appears to be related in many cases to the menopause they may result from a drying up of the normal lubricating fluid of the fascia. No detailed investigations have yet been carried out to investigate similar conditions in the region of the upper arm or round the elbow-joints, where panniculitis is also commonly found.

The Knee-joint

An abnormal fat-pad develops over the inner aspect of the knee-joints as a menopausal manifestation and perhaps precursor of ultimate osteoarthritis. It also occurs as a prominent feature of general obesity at any age, and of course in those rare cases known since 1892 as Dercum's disease (adiposis dolorosa). The appearance of these abnormal fat-pads, which may become very large and pendulous, is well known (Fig. 6). They are generally bilateral, although pain or tenderness is often confined to one side only. Posteriorly they cover the lower insertion of the inner group of hamstring muscles, being separated by a definite sulcus near the midline of the popliteal fossa from the normally present (but probably enlarged) popliteal fat-pads described above. Anteriorly they are limited by the internal aspect of the patella, but often appear to blend lower down with the infrapatellar fat-pad. There is no accompanying sensory abnormality of the overlying skin, although it is often the seat of multiple stellate or spider-like collections of thin superficial veins.

Varicose veins and local thromboses are also a common association, as is spontaneous bruising. On palpation distinct lobulation of the contained fat is easily felt, and it can be appreciated that only certain lobules—which feel more tense—are tender. If these be marked it will be found that this localized tense tenderness will affect different lobules on different days. This tenderness appears, therefore, to depend upon a condition of shifting oedema which moves from lobule to lobule. Excision of the painful fat-pad successfully relieves the pain. Microscopical examination shows only oedematous fat of normal structure and with no cellular reaction.

These pads are not to be considered as lipomata; the fat is laid down in the reticular areolar tissue between the corium of the skin and the synovial capsule, into the folds of which lobules will often penetrate and adhere. The arrangement of this areolar tissue does not seem to be as a continuous layer but in discrete sheets which when infiltrated with fat lobules in this way have the appearance each of a separate miniature mesentery. The various collections of lobules are thus in fact discrete clumps, each with its own blood supply and lymphatic system.

These medial fat-pads when they attain a large size can interfere considerably with the free function of the joint, and pain from these is quite often referred to the joint itself subjectively, although carefully objective search will reveal a tense periarticular fat lobule in the pad as its real origin. The differential diagnosis of "arthritis" and its despondent-sounding implications is all too often made in such cases, more particularly if the normally occurring "age-changes" are interpreted as radiological confirmation of this view.

Aetiology

Endocrine

The cause of the non-inflammatory oedema which is found locally to be distending the fat lobules which cause pain in the type of "fibrositis" which has been described, and in panniculitis, is unknown, although it may well be part of a more general process. It may be part of a general condition of dysfunction of fat–water metabolism, as it appears to be in those cases associated with obesity. It is this factor which accounts for those rapid and otherwise inexplicable variations in weight which occur during the dietetic treatment of the obese.

In view of the frequent association of these syndromes with the female menopause (both natural and artificial) it is tempting to suspect that the underlying cause must be endocrine. It seems to be accepted by endocrinologists that excessive secretion of oestrogens in the human will result in water retention, which largely affects the fatty tissues. This is released at a normal period, but if this does not occur the condition may become cumulative. Some reflection of this process is often seen clinically in the cases which have been described. The fault here may lie in the dysfunction of the ovarian glands or, more probably, further back in the pituitary or hypothalamus. The latter probability is strengthened by the fact that men suffer from these syndromes, more particularly the fibrositic one, quite often.

Overactivity of the pituitary and the adrenal cortex is known to be associated with pregnancy and the climacteric, and commonly results in obesity. In most cases, however, no objective evidence of endocrine disorder is to be found, and it may be surmised that the metabolism-regulating centres of the hypothalamus are disordered. This is known to result in water retention, owing to the interference with secretion of the antidiuretic factor of the posterior pituitary lobe. It is said that in these cases administration of chorionic gonadotropin will reduce the subcutaneous fat and release water, and probably the effect of this should be tried in selected cases. It may be presumed that there is no gross pathology present in the majority of the cases which are being discussed, and it seems possible that the lesion may be primarily of genetic determination. The thyroid gland has also been invoked, largely on account of its value in the treatment of certain of these cases; it is thought, however, that as this substance possesses the property of eliminating excessive tissue fluids its benefits probably do not reflect any share in aetiology.

Kling (1947), writing of what he terms "juxta-articular adiposis dolorosa" (probably a condition of panniculitis localized to the joints), states that no high degree of endocrine disturbance was found in his patients, although

B.M.R. estimates were only markedly abnormal in one-third of them. He also failed to find evidence of interstitial inflammation of sensory nerves, but he found evidence of increased tissue tension in these sites and considered it probable that the mere increase of the fat tissue causes pressure on the nerve endings and so gives rise to tenderness and pain. Where enlarged knee fat-pads were only part of a more generalized condition of adiposis dolorosa, these findings were generally found to be applicable to the other joint areas which were affected.

No generalized abnormality has been detected in the majority of our cases of panniculitis; all routine clinical tests, including the B.P. and E.S.R., were normal. Arthritis did not develop in those cases which were successfully treated for the periarticular condition. The condition is not necessarily associated with general obesity, although it may be; and when it is it tends to be more severe and widespread. Adiposis dolorosa, both in its full form and in the periarticular form described by Kling, may constitute only a variation of this entity.

Sodium Balance

Disturbances of water exchange in general betoken dislocation of sodium balance. It can be demonstrated experimentally that cells, including fat cells, swell or contract in a predictable manner in response to variations in sodium concentration in the interstitial fluid, since all cell membranes permit the free passage of water but not of sodium and chloride. The water and salt levels are now believed to be controlled largely by activity of the hormone of the posterior lobe of the pituitary and the influence of the desoxycorticosterone fraction of the adrenal cortex.

Genetic Factors

There may also prove to be a genetic factor involved, since close questioning of 50 unselected female cases of severe panniculitis revealed a history of a similar condition having occurred in the family (mostly in a mother or a mother's sister) in nearly one-half of them. This is a very high proportion, but it does not necessarily point to the disease being a familial one, since all these patients had been subjected to similar endocrine disturbance as represented by the menopause. It is perhaps more probable that it is the body type which is familial, and panniculitis certainly occurs most often in that type which Sheldon (1940) designated the endodermic somatotype.

Cold

It is striking how sudden the first onset of pain may be in some cases, leading occasionally to a wrong diagnosis for this reason alone. It has seemed that in a large number of these cases cold has been the precipitating factor, no doubt merely activating a state already in being. This is understandable in light of the observation of Bazett and his co-workers that a temperature gradient exists between the surface of the body and the deep tissues extending inwards to a depth of 2–3.5 cm. and having a temperature difference ranging from 5 to 15° F. (3–9° C.). The fatty tissues which we believe to be chiefly implicated in the type of fibrositis that has been described and panniculitis lie well within this gradient, and, since the temperature gradient—and to some extent its depth—vary in response to the cooling power of the atmosphere, these tissues must be directly affected by the external environment. These reactions are no doubt responsible for the well-attested phenomenon of weather-sensitivity in these cases.

Treatment

The pain which occurs in the two main syndromes considered in this lecture—namely, a certain type of fibrositis and panniculitis—has been shown to be due in each case to a painful distension of fat lobules within a confined space. Treatment in both will therefore be along similar lines and have the same objectives. Where these syndromes are complicated by the addition of obesity, the factor of weight reduction will also have to be faced.

The results of treatment at the present time are variable, and relapse is common. If the syndrome occurs at the menopause, however, as is not uncommon, and remission can be induced for a sufficient period to cover the completion of this process, prognosis is fairly good. The desiderata of treatment may be epitomized under various headings.

Diet.—Since fat is the site of the painful lesion in these cases weight reduction is always indicated even in patients who do not appear to be overweight. It is seldom that any improvement is noted as the result of other methods unless the body weight is falling. Restriction may be limited to starch-containing foods and to fat. Ordinarily, protein foods need not be limited.

Endocrines.—It has been pointed out that there is clinical evidence that oestrogen tends to cause fluid retention in the tissues, which is released at menstruation, and that in the menopausal patients this condition may become cumulative. Theoretically, androgens might in such cases be the proper remedy, and some recent observations tend to confirm their usefulness. In menopausal cases ordinary endocrine treatment should be tried; in patients who are not menopausal thyroid is none the less sometimes helpful, except in those cases which normally show a rapid pulse rate. In these cases thyroid extract will generally result in palpitations but no reduction in weight. There are no known drugs or hormones which will reduce pituitary activity, although such an objective would seem to be desirable.

Diuresis and Dehydration.—The type of diuresis that is required in these syndromes is that which not only increases the urinary volume but also depletes the body fluids, producing thirst. This is the type called by physiologists "obligatory diuresis." There are several substances which may be employed, and, since there is no contraindication to employing them at the same time, it is reasonable to do so. (*a*) Chemical substances which act by distorting the internal environment, such as urea. This may be given in large doses (20 g.), suspended in a little water or fruit juice after each meal, for long periods. (*b*) Chemical substances which alter the acid-base equilibrium and electrolyte balance, such as ammonium chloride. This is often given to supplement the action of mercurial diuretics, which may also be employed with benefit, and whose effect seems to be to decrease the amount of sodium chloride reabsorbed by the renal tubules. (*c*) A salt-free diet, which is effective in reducing the volume of body fluid where there is an excess. Its effect results from the removal of one of the essential components of this process, whilst restriction of water, the other chief essential, is rendered unnecessary and thirst will thus be relieved.

Physiotherapy.—Firm localized massage is used to disperse the swelling where oedematous fat lobules can be felt and are the cause of pain. It will generally prove very painful at first, and any reaction caused by this process must be allowed to die down before a subsequent treatment of this sort is administered. In time the patient will become less sensitive, and regular treatment will become possible. Previous exposure to radiant heat and infra-red rays will often increase the patient's tolerance to such treatment. Where actual herniation of fat lobules has occurred, but has not become fixed by adhesions, reduction of the herniation by this method may be possible.

Injection.—The object of local injection treatment is to disrupt the swollen fat lobule by hydrostatic pressure and so relieve the painful tension. Mere infiltration with anaesthetic solution will give temporary relief only, except in those infrequent cases in which this procedure has seemed to allow auto-reduction of a hernia, as judged by the subsequent permanent disappearance of both the nodule and the pain. The solution which we have used mostly for this purpose has been procaine 0.5% in saline. Undercutting or "teasing" with the point of the needle afterwards will complete the disruption. In cases of localized panniculitis the affected area may be thoroughly

infiltrated with an oily anaesthetic solution—such as benzyl salicylate—under pressure, with the same object in view. The fibrous compartments separating the fat lobules should afterwards be disrupted with the cutting edge of the wide-bored needle employed, to prevent recurrence.

Surgery.—Surgery has no place in fibrositis in which herniation is not a cause of the pain. Removal of irreducible fat herniae which cannot be disrupted by injection, however, remains the logical procedure, and in well-chosen cases is highly successful. These structures, however, are sometimes surprisingly difficult to locate in a well-covered person and local analgesia is essential, as the patient's subjective impressions are often necessary to ensure that it has been rightly localized. It must not be forgotten that herniations are sometimes multiple. In cases of panniculitis with obesity, persistent pain which cannot be remedied by other means can sometimes be cured surgically by undercutting the painful area. Presumably the sensory nerves responsible are cut in the course of this procedure. In two cases of persistent severe pain situated in an enlarged fat-pad on the medial aspect of the knee-joint the whole pad was excised, with permanent relief of pain, to the satisfaction of the sufferers, who had found no relief from less drastic procedures.

Several papers have been published in America during the last few years confirming the part played by fat herniae in the causation of pain in the lumbar and gluteal regions. The largest series is that of Hertz, of Cleveland. In a personal communication in 1948 he stated that since 1944 his series of such cases had reached a total of 229, 68 of which he had operated upon. At his last follow-up 62 of these had maintained complete relief from pain. Hutcherson, in a paper read before the Association for Surgery of Trauma in July, 1948, reported 42 such cases operated upon, with permanent relief of pain in 40. In England I know of no published series, although I have received accounts of isolated operations of this type which have been successful. Among surgeons who have communicated with me in this way I wish to thank Messrs. L. E. C. Norbury, S. L. Higgs, Rodney Maingot, Harold Edwards, J. C. R. Hindenach, and David Trevor.

Summary

It is thought that an aetiological classification, although desirable in the case of a "disease entity," must remain impracticable in the case of a syndrome. The classical conception of fibrositis is briefly reviewed, and a classification based upon the structure of the nodules found at the source of the pain is tentatively proposed.

Evidence is produced that the fat tissues of the body are subject to pathological variations which cause pain. This pain is generally labelled as "rheumatic" or "fibrositic." When this occurs in the neighbourhood of joints the condition is often wrongly termed arthritic.

It has been shown that several recognizable "rheumatic" syndromes can be the result of an abnormal retention of fluid by fat lobules in certain situations. When this occurs they endeavour to swell, but, being confined by indistensible fibrous tissue, they are unable to do so, and tension pain results. This oedema shows no sign of being inflammatory in nature.

In some such cases a flaw occurs in the fibrous covering, resulting in herniation of the enclosed fat lobule into an adjoining layer. Such herniations may be of three types, which are described.

The origin of this selective swelling is probably endocrine. The direct effect of cold may unmask it.

The syndromes described as being due to this abnormality of fat are: (*a*) "fibrositis," which occurs in any of the several normally occurring fat-pads which are described, and (*b*) panniculitis, which occurs in abnormally deposited fat, mostly in predictable sites. Panniculitis may occur with or without general obesity. In either case the chief sites of pain tend to remain similar. Adiposis dolorosa (Dercum's disease) would appear to be only a quantitative variation of this combination.

Treatment must be directed concurrently along several lines if success is to be achieved. Those discussed include diet, endocrines, diuresis and dehydration, local injections, physiotherapy, and surgery.

The indications for surgery are limited but tend to afford dramatic relief. These are the removal of irreducible fat herniae, and to a smaller extent removal of localized areas of painful tissue in severe cases of panniculitis.

The illustrations are from a film-strip kindly made for me by Mr. E. Wilson in the Photographic Department of the Royal Society of Medicine.

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RHEUMATOID ARTHRITIS IN THE YOUNG*

BY

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[WITH PHOTOGRAVURE PLATE]

The aetiology of rheumatoid arthritis still remains unsolved, but fresh light has been cast on the problem by a wider view of the clinical picture. The term "rheumatoid disease" has been wisely suggested (Ellman and Ball, 1948), and realization of the extensive nature of the condition, with less emphasis on the locomotor system, is a great step in the right direction. Arthritis has obviously attracted the most attention, but concentration on this single manifestation has certainly hampered search for the cause. That is why a study of the disorder can most profitably be undertaken in the young, for it is then that disease in general appears in its most acute and unadulterated form.

My remarks will be chiefly confined to certain aspects of Still's disease, which all now agree is no more than an acute form of rheumatoid arthritis in children. Further, I will try to correlate several striking features which have appeared fairly constantly in the group I have been studying with some of the rarer syndromes described by the able physicians of the past.

The study of rheumatoid arthritis, even in children, has to my mind centred too largely on the disease after it has become well established. Investigators have often not the opportunity of witnessing the onset and must rely largely on second-hand accounts from the child's relatives. In this way important developments may be missed.

Acute examples of a disease are also more likely to give information than insidious cases. I have therefore chosen for study from my case notes 20 children all of whom suffered from considerable fever and the usual general acute manifestations first described by Still. In nearly all of them careful clinical observations were made early in the disease, if not from the onset; subsequently the progress of the children was followed for many years, sometimes into adult life.

The account deals particularly with the early stages and general aspects of the disease; following the principles already expressed, arthritis will receive only short consideration.

*Read in opening a discussion in the Section of Rheumatology at the Annual Meeting of the British Medical Association, Harrogate, 1949.

Mode of Onset†

This is more or less abrupt and could often be dated to a day. In most instances there was considerable fever and the disease began with *arthralgia*. At first there was little obviously wrong with the joints until attempts were made to move them, which were painful. Sometimes they were puffy and, though somewhat stiff, movement was not greatly restricted. Later definite *arthritis* set in, but at this early stage it was usually migratory, flitting from joint to joint and lasting only for a few hours (see Chart I). Redness of the surrounding skin has been observed in some instances, and the glistening appearance, swelling, and pain so resembled gout that blood uric acid levels were estimated but were always found to be normal. Sometimes the onset was like that of rheumatic fever, but failure of adequate salicylate therapy soon proved this diagnosis to be wrong.

In a few cases joint manifestations were minimal or entirely absent, and the main clinical features were listlessness, anorexia, adenitis, an unexplained fever, and a rash. The diagnosis then generally inclined towards glandular fever or brucellosis, but this was not supported by laboratory tests.

Adenitis.—This constantly occurred at some period, but with few exceptions could not be counted as one of the earliest features. Once present the enlarged glands persisted throughout the acute phase and disappeared only if the child made a good recovery. When a more chronic form of rheumatoid arthritis developed glandular enlargement continued until the disease became "burnt out."

CASE	SEX	AGE AT ONSET	RASH	SPLEEN	LIVER	JAUNDICE	INITIAL LEUCOCYTOSIS W.B.C. per cmm.	LATER LEUCOPENIA	PERICARDITIS	FEVER: IRREG. CONT.	FEVER: PERIODIC	RESULT
1 C.B.	M	10/12	+	±	++	SLIGHT	18,000 P. 87	1500 P.15 L.85	P.M.	+	±	* LEUKÆMIA DEATH
2 T.D.	M	2 4/12	+	±	–	–	24,000 P.64	–	–	+	–	ARTHRITIS AMBULANT
3 J.C.	F	6 9/12	+	+	–	–	31,000 P.92	–	–		+	COMPLETE RECOVERY
4 A.B.	M	10 6/12	–	–	–	–	17,000 P.72	–	–	+ →	±	ARTHRITIS
5 W.C.	M	5 2/12	+	–	–	–	22,000 P.78	–	–		+	RECOVERY
6 E.L.	M	3	–	–	–	–	14,400 P.66	2,900 P.53 L.44	P.M.	TERMINAL		* DEATH
7 R.H.	M	6	+	–	–		22,800 P.77	–	+		+	ARTHRITIS
8 I.M.	M	7 6/12	+	–	–	–	20,000 P.80	–	–	+ →	+	MIGRATORY ARTHRITIS
9 M.H.	M	2	+	–	–	–	29,000 P.82	–	–		+	DEATH (GOLD) *
10 K.S.	M	4 3/12	+	+	+	–	12,700 P.84	–	–	+ →	+	MIGRATORY ARTHRITIS
11 W.H.	M	2 7/12	+	++	+++	SLIGHT	19,200 P.57	–	–	+ →	+	NO ARTHRITIS HEPATIC CIRRHOSIS
12 G.H.	M	6	+	+	+ later	+	21,200 P.82	–	–		+	SLIGHT ARTHRITIS DEMENTIA
13 M.H.	F	3 9/12	+ later	+	+	–	NORMAL	–	?(PLEURISY)	+ ←	+	ARTHRITIS
14 A.B.	M	9/12	–	+ later	+ later	–	19,000 P.75	–	?(PLEURISY)	+ ←	+	SEVERE ARTHRITIS
15 A.B.	F	2 2/12	+	+ later	+ later	+	28,200	3400 P.71 L.24	+		+	SLIGHT ARTHRITIS RECOVERY FROM LEUCOPENIA
16 M.C.	F	9	–	±	+	–	17,500 P.72	–	–	+ ⇄	+	SEVERE ARTHRITIS
17 J.C.	M	4	+	+ later	–	–	22,600 P.87	–	+	+ →	+	ARTHRITIS AMBULANT
18 E.M.	M	5	+	+	+ later	–	16,500		+	+ ←	+	RECOVERY
19 S.D.	F	3	+	–	–	–	13,900 P.76	–	–	±		ARTHRITIS
20 D.P.	M	3 9/12	–	++	+	–		3,000 P.3 L.97	–		+	* LEUKÆMIA DEATH

Table incorporating the chief clinical points referred to in the text. Adenitis was an early feature in all cases. Subcutaneous nodules appeared only in Cases 8 and 12 at a later stage. Gold therapy in Case 9 provoked a severe and fatal reaction almost at once.

Splenomegaly.—This was not prominent at the beginning and was found to any degree in only just over one-third of the cases. Later enlargement of the spleen became more obvious.

The Rash.—This appeared in 15 children, usually at the onset, and varied in intensity more or less with the temperature. At a later stage it vanished altogether, and that is no doubt why it has not been greatly stressed in the literature. The rash had a fairly widespread distribution on the limbs, the trunk, and occasionally the face. It was sometimes urticarial at first but without the usual pronounced irritation; soon it became maculo-papular and was often migratory. With the associated adenopathy it is not surprising that cases have been wrongly suspected of being rubella. Rarely the macules fused into a widespread erythema; occasionally petechiae appeared and the capillary fragility was found to be raised.

Fever.—In his original description Still stated that the fever might be continuous or might occur in bouts. Both types were seen in 16 of the 20 patients: usually a prolonged intermittent or a high irregular continuous temperature in the opening stages, giving way to periodic febrile attacks later (see Charts I and II). The periods of pyrexia were not so clearly defined as in Hodgkin's disease or melitensis, or undulant, fever, but careful analysis clearly showed their phasic character, which varied considerably from case to case and from bout to bout (see Charts I–III). In fact, the temperature in some children never remained normal for long; nevertheless the "switchback" appearance of the curves was typical (see Chart IV).

Both joint involvement and the rash tended to lessen or to vanish completely when the temperature dropped. Adenitis and a raised B.S.R., on the other hand, persisted throughout the acute and active stages of the disease. During the bouts of fever the child was very miserable, obviously ill, and in pain. Improvement followed the fall in temperature, but the clinical condition slowly deteriorated with each successive rise. Eventually the febrile attacks ceased and a typical clinical picture of varying degrees of rheumatoid arthritis remained (11 cases). Complete recovery with no obvious residual arthritis was the final result in three (cf. Hench, 1949), death occurred in four from causes which will be discussed later, and in two the acute stage has not yet terminated.

Blood Picture.—With any prolonged fever anaemia is to be expected, and it was not surprising to find before long a reduction of the red cells to between three and four million per c.mm., with a corresponding drop in the haemoglobin. More significant was the pronounced leucocytosis, almost invariably present at the beginning of the disease (18 out of 20 cases). Counts between 17,000 and 30,000 white cells per c.mm. were commonly registered, with a pronounced polymorphonuclear cytosis in the region of 80 to 90%. Very soon, however, the number of white cells returned to normal and leucocytosis was seldom repeated (see Table and Chart IV).

On rare occasions and at any stage the disease appeared to become too much for the bone marrow. Severe leucopenia then set in. Both neutrophils and lymphocytes might be equally affected, but agranulocytosis has also been observed (Plate, Figs. 1 and 2), and in two cases the final clinical picture and bone-marrow biopsy were indistinguishable from that of *lymphatic leukaemia* in an aleukaemic phase (Cases 1 and 20). Leucopenia developed in four cases—three patients died (Cases 1, 6, and 20), and one recovered rapidly and soon had a normal blood count (Case 15).

Other Clinical Features and Complications

Pericarditis is a well-known manifestation, and occurred in six of the series. It may in fact precede all other manifestations and be mistaken at first for acute rheumatic pericarditis (Chart III). Doubt arises when the illness does not follow the usual course and the myocardium is not found to be greatly involved. Sometimes the physical signs are not very obvious; pericarditis is then masked by the acute febrile disturbance and is observed only at necropsy (Cases 1 and 6).

†The following section should be read in conjunction with the accompanying Table, in which the main features of the 20 cases are summarized.

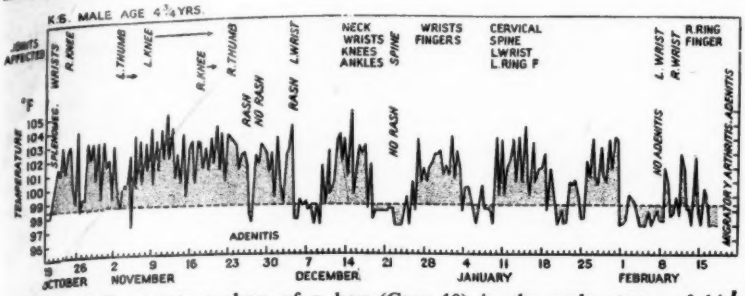

CHART I.—Temperature chart of a boy (Case 10) in the early stages of his disease, showing at first an irregular continuous form of pyrexia which subsequently became phasic in character. The migratory nature of the arthritis is also well demonstrated.

Pleurisy and pneumonitis are commonly associated with the pericarditis, but occasionally they may occur on their own (Cases 13 and 14) (cf. Ellman and Ball, 1948).

Jaundice figured as an occasional complication in Still's original account. In the present group it appeared in four cases and the liver became enlarged in nine. In one remarkable instance (Case 11) the liver and spleen reached well below the umbilicus, and laboratory tests at the time demonstrated impaired liver function. After a year's illness all signs of rheumatoid arthritis vanished; the boy appeared well and active, but was left with an enlarged spleen and *cirrhosis* of the liver (see Chart II and Plate, Fig. 3). Amyloid disease has been described (Imrie and Aitkenhead, 1939), but it was never demonstrated during life or after death in the present series.

Only one account of *encephalitis* as a complication could be found in the literature (Spühler, 1941). This was in three adults with Still's type of acute rheumatoid arthritis. In one of my cases (No. 12) convulsions and prolonged coma suddenly developed with pneumonia, pyelonephritis, cardiac failure, and transient hyperpiesia (B.P. 200/120) as additional features (see Chart IV). The boy recovered from all these, but has been permanently aphasic and demented ever since. The aetiology of all this is obscure. Polyarteritis nodosa has lately been much debated in relation to rheumatoid arthritis, and the question naturally arose in this case, but the condition was not confirmed by muscle and vessel biopsy.

Among other early features in the series were excessive sweating, rapid muscular wasting, and generalized bone rarefaction. Later hirsuties, particularly round the labia, was often noticed in quite young girls and is difficult to explain. One other factor should be mentioned: in a few isolated instances the onset of the disease followed within a few hours of some severe trauma or shock, such as a fall down a deep chalk pit, with concussion, or sudden accidental immersion in cold water. Such occurrences may be purely fortuitous and have no bearing on the problem, but they may have some aetiological significance. Similar happenings are well known to anyone who has had wide experience of juvenile rheumatism, and serious relapses have been observed in children with acute rheumatic carditis following fractures, after surgical operation, or subsequent to a long drive in an ambulance (Bland and Duckett Jones, 1935).

Relationship to Other Syndromes

Henry Cohen (1948) in a chapter on "the rarer arthritic syndromes" has picked out one or two groups which seem to be interrelated. Some of them bear a striking resemblance in part of their clinical course to the acute form of rheumatoid disease just described. In his description of *intermittent hydrarthrosis* Garrod (1910) refers particularly to the associated skin rashes, which might be urticarial, erythematous, or haemorrhagic. Although the knee-joints were usually alone affected, others have observed a migratory arthritis. The disorder differs, however, from the disease we are considering in its more benign course and in the greater regularity of its periodic outbursts. In Garrod's cases the condition was mostly superimposed on damaged joints; in mine it was often the precursor of permanent arthritis. It is interesting to find that Garrod also mentions injury as a precipitating cause.

Pyrexia is not a great feature of intermittent hydrarthrosis; when present it coincides with the joint swellings and exhibits a fairly regular periodicity. More severe fever occurred in an allied disorder which Solis Cohen (1913) called "angioneural arthrosis." About 40% of his cases were febrile, and in a number of them the pyrexial attacks had the same phasic character.

Comparable also to a few of the cases in my series was the erythematous or gouty appearance of the joints during the attacks. More or less the same clinical picture was described by Hench and Rosenberg (1944) in a condition they term "palindromic rheumatism," which seems to be a milder form with no effect on the blood sedimentation rate, no fever, a normal blood count, and little tendency to chronic arthritis, but with widespread nodule formation. Palindromic rheumatism has also attracted the attention of Parkes Weber (1946), who finds a common thread running through all these separately described syndromes, including also those of Schlesinger (1899) and Kahlmeter (1939). In many respects their course differs considerably from that of Still's disease in its usual form, but several striking points of resemblance can be found.

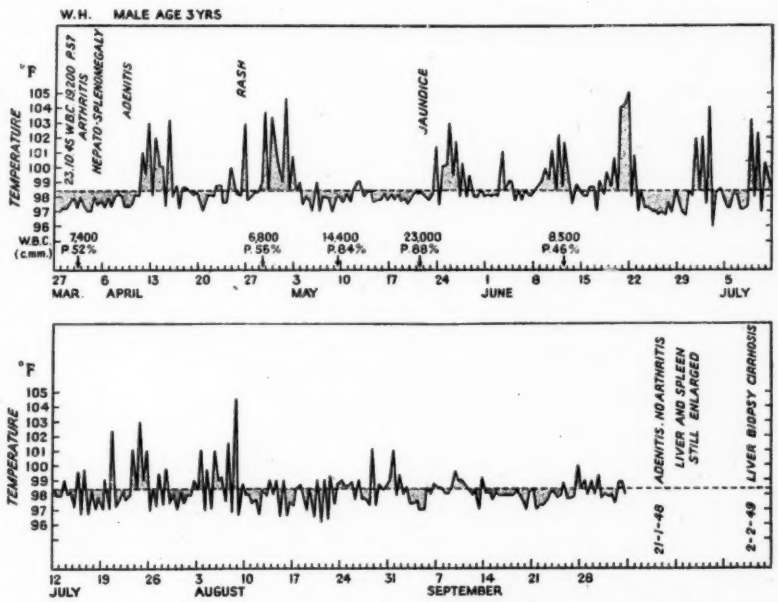

CHART II.—Chart of a boy (Case 11) five months after the onset. Short periodic bursts of pyrexia are shown. Leucocytosis, present at the onset, is no longer evident; the temporary recurrence later was associated with jaundice. There was complete recovery from arthritis, but cirrhosis of the liver developed (see also Plate, Fig. 3).

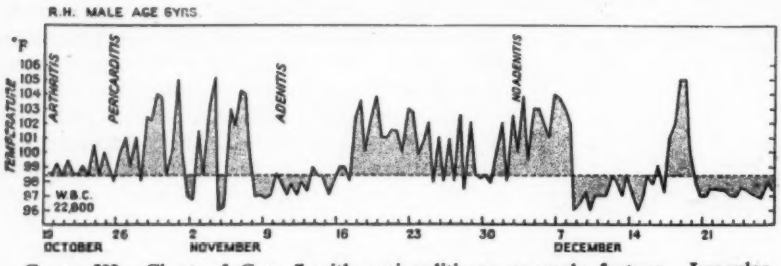

CHART III.—Chart of Case 7 with pericarditis as an early feature. Irregular bouts of fever are again obvious.

Precursor to Leukaemia

The rare termination of Still's disease in lymphatic leukaemia, which I have discussed, raises all kinds of possibilities. Preceding this there may possibly be a stage of undifferentiated leucopenia (panleucopenia), a complication of acute rheumatoid arthritis first described by Felty (1924). In his syndrome the neutrophils were not specially picked out and the low white cell count persisted for many months without the patient being in great distress or the joint affection severe. Felty remarked upon the associated pigmentation and the resemblance of his cases to those described by Banti. The glands and spleen were enlarged but not the liver, although in one case some liver impairment was suspected.

There are also one or two accounts of agranulocytic leucopenia in rheumatoid arthritis, not attributable to any specific form of therapy (Collins, 1937; Blechmann and Carcanagues, 1940). None of these cases proceeded to leukaemia, but this followed in one patient described by Türk (1938).

It seems as if the severer cases of Still's disease are in danger of developing leucopenia. From this the patient can occasionally recover, but the condition is more likely to proceed to agranulocytosis and in rare instances degenerate into leukaemia. Further research by repeated bone-marrow biopsy during the course of the disease is required in order to discover the exact sequence of events.

Discussion

Recently some form of allergy has again been suggested as the basis of the pathological process in rheumatoid arthritis, and in support of this two rare conditions—lupus erythematosus and polyarteritis nodosa—have been repeatedly quoted. Both are thought to be allergic disorders and to bear a certain resemblance to acute rheumatoid disease. This is suggested by the pathology of *lupus erythematosus*, in which the collagen tissue is chiefly affected by fibrinoid changes (Klemperer, 1948; Humphreys, 1948). The clinical features—fever, glandular swelling, arthritis, anaemia, leucopenia, and occasionally pericarditis and diffuse or focal cerebral disease—are also significantly similar to the symptomatology we have been considering. Focal myocarditis, another frequent lesion in lupus erythematosus, might be taken as further evidence of a common pathology, particularly by those who consider rheumatic fever and rheumatoid arthritis to be associated.

The morbid anatomy of *polyarteritis nodosa*, on the other hand, does not seem to bear such a close relationship, but its clinical course is somewhat similar, particularly the arthritis, fever, sweating, prostration, tachycardia, skin eruptions, and occasional involvement of the pericardium (Schlesinger and MacCarthy, 1949).

Glomerular nephritis, which is common in these two rare disorders, is not a feature of Still's disease. The link here is perhaps more with serum disease produced experimentally in animals and resulting in a number of lesions which are also found in human serum disease, nephritis, polyarteritis nodosa, rheumatic fever, and rheumatoid arthritis.

Comparison of Still's disease with these two rare disorders, whose character has only recently become clearer, and with syndromes of the past in which an infective factor has always been in doubt does seem to favour some allergic basis as the underlying cause. Its exact form is still most obscure. No one has yet discovered the nature of the antigen, explained how it produces a prolonged disease with progessive inflammatory lesions, or discovered what predisposes certain individuals and allows others to escape. Possibly some organism, normally innocuous, may be living in harmonious symbiosis with its human host and suddenly become pathological when the balance is somehow deranged by stress or trauma. This might explain the occasional effects of shock, to which attention has already been drawn.

The dual role, partly infective and partly sensitizing, that bacteria can play in the development of disease is well known. The tubercle bacillus in tuberculosis and the streptococcus in nephritis and in purpura are good examples. So far no specific infection has been constantly found to precede the onset of rheumatoid arthritis, but an infective factor is strongly suggested by the pronounced polymorphonuclear leucocytosis which was revealed in the early stages of nearly all the cases under review. Leucopenia and agranulocytosis might possibly be the detrimental effect on the bone marrow of repeated allergic reactions.

The changing character of the fever from a more or less continuously high level to an irregular periodicity might also conceivably be regarded as the successive result of an infective and an allergic process.

Mesenchymal lesions in the skin, lymph nodes, joints, heart, lungs, and spleen result from the experimental

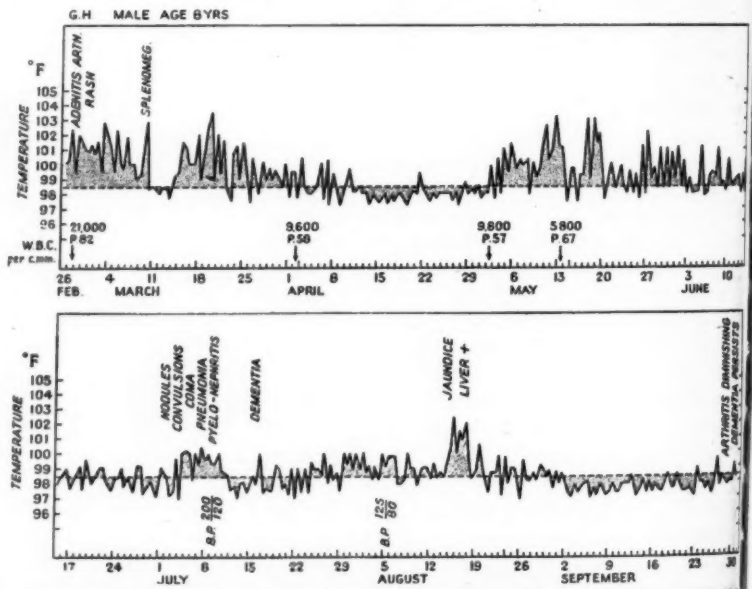

CHART IV.—Showing the "switchback" fever chart of Case 12, with encephalitis and dementia as complications. Initial leucocytosis occurred, but did not persist for long.

intravenous injection of soluble antigens (horse serum) into animals, while somatic antigens (whole bacteria) produce their effect mainly in the lungs, liver, and spleen (Ehrich *et al.*, 1949; Gregory and Rich, 1946). Most of these are regions commonly involved in Still's disease, and, as has been shown, the lungs, the heart, and the liver may not escape.

Conclusions

Study of the most acute cases of Still's disease emphasizes the widespread nature of the lesions. Although the majority of cases proceed to progressive arthritis, some patients may recover completely. Characteristic types of pyrexia can be recognized at different stages, and significant effects on the blood picture have also been observed. In rare instances the clinical picture may change from Still's disease to leukaemia. The close resemblance of Still's disease, particularly at the onset, with certain rare syndromes of an allergic nature suggests that rheumatoid arthritis may have a similar aetiological basis. Should this be so, some form of primary bacterial invasion seems to be likely in the course of events.

I am indebted to several of my medical colleagues for allowing me to study their cases, and to Dr. Bodian and Mr. Martin for the histological sections and microphotographs reproduced in the photogravure plate.

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The current issue of *Planning* is the three-hundredth in the series which PEP began some 17 years ago. It looks back on the early history of PEP between its foundation in 1931 and the outbreak of war. A brief account of its origin mentions the men who were most concerned with the development of the group. In the early days PEP was given some special grants by the Rockefeller Foundation, but over the period 1933–9, 12 major and two minor reports and more than 150 broadsheets were published on a budget of no more than £56,000 for these seven years. There is a brief description of the *ad hoc* working groups set up by PEP, and an attempt is made to assess its contribution to contemporary knowledge and ideas. The first major report to be prepared by the Civic Division of PEP was that on *The British Social Services*, and this was followed in December, 1937, by the *Report on the British Health Services*. Three groups which did important work during the war were the Social Security Group, the reconstituted Health Group, and the Population Policies Group. The report on *Population Policy in Great Britain*, however, was not published until April, 1948.

PSYCHOGENIC RHEUMATISM

BY

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In a large hospital to which patients are referred suffering from "rheumatism" considerable diagnostic experience is needed in order to classify the various conditions included in this omnibus term. There is no agreement at present on the scope of the term; it is customarily taken to indicate a group of painful disorders of the locomotor system. It is obvious that a large number of syndromes come under this heading. While the arthritides, neuralgias, and other somatic conditions as a rule present straightforward signs and symptoms, patients are continually encountered whose experiences do not seem to fit in with any recognized syndrome and in whom physical signs are absent or minimal. In such cases one may be tempted to regard the patient as suffering from pain which is primarily of psychogenic rather than organic origin. If this hypothesis is correct, it is plain that rheumatic disorders of this type call for particular care in diagnosis and for treatment more far-reaching than analgesics and physiotherapy.

In recent years there has been a growing disinclination to accept all rheumatic complaints as somatic in origin. Even before the war one of us had coined the term "polyalgia syndrome" to describe the symptoms of which some patients complained. In England, Ellman *et al.* (1942) studied 50 patients with "fibrositis": 23 had no physical signs, although they had been diagnosed as suffering from somatic disease; 25 showed hysterical tendencies. An interpretation of the rheumatic complaint in psychological terms was given by these workers. An important contribution was made by Flind and Barber (1945); many of the subjects investigated by them were found to be suffering from psychiatric rather than organic states. Hench and Boland (1946) gave a full description of psychogenic rheumatism in a paper on the incidence of rheumatic disease in the United States Army; this condition was the third most common diagnosis made in their series of 800 patients.

If psychogenic rheumatism is to be accepted as an entity both by those who diagnose and treat rheumatic disease and by psychiatrists, our concept of it needs clarification and acceptable diagnostic criteria must be established. This demands close collaboration between clinician and psychiatrist: it is the purpose of this paper to show the results of such an endeavour.

Fifteen patients regarded by the clinician as suffering from psychogenic rheumatism were referred for detailed psychiatric examination. In each case the patient was first examined by routine clinical methods. The haemoglobin and sedimentation rate were estimated. The group contained 14 women and one man; the distribution by age was: 15–29, four; 30–39, three; 40–49, four; 50 and over, four. The preponderance of women in this group was in accord with the assumption that psychogenic rheumatism in clinical practice is considerably commoner in women. A control group of patients suffering from a painful disorder of which the cause was known was investigated by the same means. This group consisted of subjects of the same age and sex distribution, and broadly of the same social class. The diagnoses were: osteoarthritis (3),

brachial neuralgia (3), fracture of a joint (3), cervical rib (1), periarthritis (1), lesions of an intervertebral disk (2), rheumatoid arthritis (1), alveolar abscess (1). The psychiatric examination was conducted along three main lines—clinical interview, psychometric examination by two projective methods (Rorschach test and Thematic Apperception test—T.A.T.), and appraisal of information given by a relative, employer, or friend.

Results

A. Form of Rheumatic Complaint

Expression of Complaint.—Brief and consistent terms ("sharp," "sore," "aching"): controls, 10; patients, 3. Compound or unusual terms ("twisting," "heaviness," "burning," "stinging and stabbing," "shooting and numbness," "terrible," "hard to describe"): controls, 5; patients, 12. The patients often seemed to be trying to put into words an indescribable sensation. The impression to the observer is frequently that of a mystical or ecstatic experience, with an undercurrent of satisfaction, which recalls Helene Deutsch's (1947) elaboration of masochism as a primary feminine trait. Terms may be used which convey the description of a mental state rather than of a physical stimulus—e.g., "irritation." The psychological precipitants are at times stated quite plainly—e.g., "boredom causes irritation."

Site of Discomfort.—Localized: controls, 11; patients, 3. Widespread: controls, 4; patients, 12. In the majority of the control group discomfort was confined to the area of the lesion or to an area related to it. The distribution of discomfort in the test group was commonly very extensive—e.g., neck, spine, knees, shoulders, calves, hands. The phrase "pain all over" was employed by several patients.

Factors Influencing Severity of Discomfort.—In the controls the most common aggravating factor was touch or movement of the affected part: 11 cases. Among the patients the following were mentioned: work, 4; relaxation, 4; mood, 2; noise, 1; heat or cold, 2; doubtful, 2. Relief of distress in the control group followed rest in 10 cases and treatment in four. Of the patients, seven did not admit relief with any activity or form of treatment; in the remainder some relief occurred with rest or exercise, a hot bath, or quiet. Analgesic drugs produced improvement in six out of seven control subjects in which they were tried; out of eight patients, none noticed any effect from these drugs. Two control subjects considered that the complaint was influenced by their state of feeling, as compared with six patients. Variations in the severity of discomfort throughout the 24 hours, or from day to day, showed no great differences between the groups. The effect of climatic conditions—cold and damp—was similar in both groups. Degrees of disability from slight to total were noted in six control subjects; only one patient suffered any disability, and this was slight.

Associated Symptoms.—Depression, tension, irritability, poor sleep, or phobias were present in 14 of the patients and in five of the controls. In two of the latter the nervous symptoms were considered to be secondary to the somatic disorder.

B. Circumstances of Onset

Two of the controls and 13 patients became aware of the onset of the disorder in circumstances associated with emotional tension. In the remaining 13 controls the onset of illness appeared to be fortuitous. In the majority of the test group the rheumatic condition could be shown to have a comprehensible meaning in relation to the patient and her life situation.

C. Psychiatric Status

Clinical Examination.—In 10 of the control group no significant psychiatric abnormality was found. In the other five the physical condition was accompanied by nervous symptoms:

The first of these subjects complained of worries, fears, tension, and fatigue. It was considered that her conditions of housing were producing anxiety and frustration, which increased the disability due to her organic state (osteoarthritis). In the second, depression, lack of energy, and poor sleep appeared about one month after the onset of brachial neuralgia and seemed to be a consequence of pain and limitation of activity.

In the third, a right-sided cervical rib had begun to cause symptoms a few days after a fall on the right hand. On examination the patient also admitted to shakiness, depression, disturbing dreams, and fear of the dark and of open spaces. These were discovered to accompany sexual maladjustment with dyspareunia and fear of pregnancy. They appeared unrelated to the rheumatic complaint.

In the fourth subject pain from periarthritis of the right shoulder was accompanied by fatigue, headache, and insomnia. The shoulder condition followed a fall a few months before the time of examination; the other symptoms had been present over many years and were the expression of a lifelong neurotic state.

The fifth subject, with a twelve-months history of rheumatoid arthritis, complained of lassitude, depression, and disturbed sleep. She gave a history of previous nervous illness. In view of the common observation that rheumatoid arthritis may become manifest after emotional trauma, the circumstances of onset in this patient were carefully reviewed. No significant precipitating event was found, and the nervous symptoms were considered to be secondary to the arthritis.

In only one patient of the test group were there no associated nervous symptoms. With two possible exceptions the rheumatic condition in every patient in this group formed one component of a reaction pattern which was also expressed in disturbance of affect and behaviour. The pathogenesis of the condition in each patient is considered in more detail below.

Psychometric Findings.—(*a*) *Rorschach test:* This is a sensitive indicator of personality disturbance. Interpretation of records is a skilled task and cannot be reduced to a quantitative scoring method. For the purposes of this investigation the accompanying table of incidence of certain Rorschach signs of maladjustment was drawn up; it shows a greater degree of disturbance in the test group than in the control group taken as a whole. If the two control subjects with neurotic features on clinical examination are excluded the difference becomes more pronounced. More patients than controls showed evidence of difficulties in the emotional field: domination by emotional impulses or a morbid fear of and withdrawal from situations likely to arouse them. "Blocking"—complete failure to handle adequately such situations—was more often found. Awareness of alien forces within the personality of a sinister and threatening nature was commoner in the patient group.

Results of Rorschach Test

	Responses < 15 or > 70	No Colour Response	Colour Response C Only	Rejections Other Than IX	Last 3 Cards: Under 30%	Last 3 Cards: Above 45%	Last 3 Cards: Total	Content Categories Under 5	P. Responses Under 4
Controls	4	4	—	5	6	—	6	6	7
Controls, without neurotic subjects	2	2	—	4	6	—	6	4	5
Patients	11	6	2	8	8	2	10	10	11

Signs of sexual maladjustment occurred more often. The various effects of anxiety on the test performance were shown to a considerably greater extent in the controls than in the patients' records.

(*b*) *Thematic Apperception Test* (selected series of cards): The patients showed in general a greater preoccupation with themes of violence, injury, crime and punishment, murder, and suicide. For example, one at least of the cards was interpreted as showing a murder by six of the patients, as against three controls; as showing a suicide by four patients, as against one control; and as showing some form of violence by five patients, as against one control. Themes of guilt after wrongdoing were also more frequent in the patients' records.

The material from the Rorschach and T.A.T. records will be used at a later date to build up a formulation for the pathogenesis of illness in the test group.

D. Physical Status

The control group showed the physical signs appropriate to the physical disorder from which they were suffering. The complaint was in general consistent in its expression and the discomfort was localized to one, usually anatomical, body area.

The test group complained in the main of diffuse and severe discomfort, inconstant in site, in degree, and in nature. The terminology used to express the complaint was often bizarre. The distribution of the discomfort did not correspond to that of a recognized physical disorder. Physical signs of a skeletal abnormality were absent. To say, however, that no physical signs can be elicited is not usually correct. A typical reaction to examination was found. Tenderness was often diffuse and due to skin hyperaesthesia. The patients found relaxation difficult, and muscular tension had often to be overcome before full joint movement could be demonstrated. Deep reflexes were exaggerated. In several cases the patient took the examiner's hand and applied it to the painful portions of her body.

X-ray examination and sedimentation rate estimation in the test group showed no abnormality.

Pathogenesis

The absence of physical signs of organic disease and the clinical characters of the complaint, especially the distribution of the discomfort and the significance of the complaint in the patient's total reaction, place this type of rheumatism in a class apart from the hitherto recognized rheumatic disorders. Its formulation as a "psychosomatic" condition cannot, however, be considered as adequate unless the morbid physiology underlying the physical symptoms can be articulated with the psychodynamics of the patient's emotional state.

In the test group a tentative formulation of the pathogenesis could be arrived at in 13 of the 15 cases. A fuller account embodying the psychometric material and further physiological data will be published later; a summary of each case, necessarily simplified, is here given.

Miss A., aged 24, is described as "fussy, upright, proud; holds herself very upright." Mother: rheumatism; nervy, quick-tempered. Mother died in 1943; in 1945 father began to carry on a liaison with another woman, who drank. Patient objected to this, and there were quarrels, after which she felt very disturbed and tense. Her feelings "seem to bottle up inside." Onset of generalized pains, mainly neck and spine, in 1945. Pathogenesis: somatic expression, perhaps in muscular tension, of strong moral resentment of father's behaviour.

Mrs. B., aged 50, was nervous and timid in childhood; illness diagnosed as chorea. Nail-biting and enuresis until 11 years. Home unhappy. Marriage unsatisfactory; husband has "bad nerves" and accuses her constantly of infidelity. "Prolapse of womb" in 1946; after this she was convinced that "something was wrong inside" which had not been discovered. Pain in the right side and back began at this time, accompanied by hysterical outbursts. Pathogenesis: nosophobia.

Mrs. C., aged 45. Father: violent, unstable. Mother: nervy. As a child patient was timid and nervous and afraid of father. Marriage happy; wanted large family but had only one child. Operation at 30 for sterility; at 42, uterus and one ovary removed. Very distressed by this; felt inferior to other women. Backache began after the operation. Rorschach: gross disturbance by a sinister fantasy life, with aggressive and sadistic trends. Pathogenesis: somatic expression of self-punitive trend at her failure to bear children.

Mrs. D., aged 39, is an unstable, dull, shallow person who nevertheless has considerable determination and can usually get her way with people. Married during the war and left her husband next morning as she could not tolerate sex relations. Felt strongly the need for home and security. Onset of pain in back and perineum while living alone in London. This resisted all treatment until she found a congenial job with friendly people. Pathogenesis: somatic expression of intense self-concern and need for affection.

Miss E., aged 27. Father: rheumatism; bad temper. Mother: rheumatism; irritable. Patient a diligent, conscientious, active person. Is compelled by circumstance to do a good deal of housework as well as her job, and resents it. Both parents are difficult and obstinate, and there are continual family quarrels. Onset of generalized pains after an increase in household duties on her sister's marriage. Pathogenesis: somatic expression of suppressed resentment of her parents' attitude and demands.

Mrs. F., aged 42, was nervous and timid as a child; enuresis till 12 years. Regular dysmenorrhoea; after marriage, disinclination for sex. Onset of pain in rectum while on evacuation from London during the war; this was relieved by injection for haemorrhoids, but she was told that the "neck of the womb was out of order." Became convinced that she had some disease of the uterus, probably cancer; pain in rectum returned, with backache. Pathogenesis: nosophobia.

Mrs. G., aged 53. Father: rheumatism "all over"; irritable. Mother: rheumatism. Patient nervous and worrying as a child. Family unhappy. All her life has had to look after invalids; her husband died of tuberculosis after a long illness. Had one child which lived only two weeks; pain in the back at this time. Had to take care of a sister with Parkinsonism who did not appreciate what was done for her; exacerbation of rheumatism at this time. Depressed; considers her life is empty. Pathogenesis: somatic expression of anxiety with guilt over her aggressive impulses towards sister.

Mr. H., aged 23, was wayward and delinquent as a boy; preferred sport to work. Served in R.A.M.C. during the war, much of the time abroad. Found this "nerve-racking"; longed for home. Onset of pain in left chest during a football match, probably from a minor injury, in Singapore after return from home leave. After demobilization, dissatisfied with civilian job and living conditions. Pathogenesis: prolongation of physical symptom due to frustration and discontent.

Mrs. I., aged 42. Mother: rheumatism; very concerned over children's health. Patient is married and has four children, who are a constant worry to her lest they fall ill or are injured. Never lets them out of her sight, but is impatient if they resist her—"would like to take a knife to them." Onset of backache during a depressive illness. T.A.T.: preoccupation with death and suicide. Pathogenesis: somatic expression of tension from markedly ambivalent feelings towards children.

Mrs. J., aged 33. Father: backache, nervous. Mother: "curved spine." Patient always wished to do well in business and was happiest when managing a public house. Has always been irritable, tense, and unable to relax. Marriage fairly satisfactory, but she had dysmenorrhoea, distaste for sex relations, delayed conception, and vomiting in pregnancy. She sleeps badly, and while half awake sees vivid pictures of death

and injury. T.A.T.: preoccupation with and fear of sadistic impulses. Onset of pain in back, legs, and hands during last pregnancy. Pathogenesis: somatic expression of discontent from thwarted masculine aims and ambitions.

Mrs. K., aged 32. Father: rheumatism in head, arms, shoulders, knee; "always rubbing himself"; "nerves in the head." Patient "takes after father very much." Onset of aching pain in back, arms, and legs while living away from London during the war. No immediate source of emotional tension found. Patient very preoccupied with her complaint and sure she is going to be crippled with rheumatism, as her father and aunt were. Probable pathogenesis: nosophobia.

Miss L., aged 19. Father died when patient was 11. Brought up by mother, with whom she has always got on badly. Patient described as "hysterical; flares up easily." Both she and her mother have violent tempers. Patient says she wants to leave home, yet is clearly very dependent on mother. Has one boy friend, with whom she quarrels continually. They became engaged, hoping that this would ease things; the quarrels ceased, but the patient developed pain in shoulders and arms, with odd sensations in the limbs. Pathogenesis: somatic expression of anxiety from two main conflicts—over dependence, and over relation to fiancé; with utilization of illness as a means of avoiding marriage.

Mrs. M., aged 52. Father: ill-tempered and unstable; left home. Patient strong and active; reared in a convent. Marriage difficult; husband a chronic invalid who died, leaving her with five young children. She had to work to support the family. Onset of pain in back and limbs after a confinement in her early thirties. Repeated mild attacks of depression, ascribed to worry of looking after children. T.A.T.: marked preoccupation with theme of injury and reparation as between mother and child. Probable pathogenesis: somatic expression of guilt over care of children.

Miss N., aged 59. Father: nervous, temperamental. Mother: rheumatism. Patient has energetic and forceful disposition, with strong intellectual and social ambitions, which have never been realized. At about 24 she began to suffer from insomnia with "visions," in which she could see herself as a crowned queen; with this, she felt irritation under the skin. At about 50 pain began in knees, ankles, and fingers, worst during the night, when she cannot sleep. T.A.T.: preoccupation with and fear of sadistic impulses. Pathogenesis: somatic expression of suppressed resentment from thwarted ambition, precipitated by menopause.

Miss O., aged 43. Mother: fainting attacks; chronic fatigue; dominating and strict with her children. Patient active, vigorous, ambitious, and successful; reached her goal—to be a head mistress. Onset of backache during the war, at a time of especial stress while she was concerned over danger to her mother from the air raids. Had to give up her career to look after mother because of illness. Pathogenesis: somatic expression of anxiety from conflict over relation to mother.

Diagnosis and Treatment

The principal diagnostic features of psychogenic rheumatism appear to be in (1) expression of complaint; (2) area of discomfort; (3) failure to conform to an established organic syndrome; (4) setting of the illness in relation to the patient's life situation; and (5) lack of response to physical treatments.

The treatment and disposal of patients regarded as suffering from psychogenic rheumatism has always provided a real problem to the clinician. It is part of the syndrome that they do not respond to treatment which is limited to physical measures. It seems clear that to these patients their suffering is something very personal and valuable; it makes them important, interesting, and worthy of sympathy, and is a protection against an unkind world. To treat them with passive physiotherapy is to acquiesce in their own diagnosis of disease and to allow it to take a deeper root. Such treatment usually gives temporary relief but does not cure; after a time it may come to be indispensable to the patient. Physiotherapy which is painful may remove the symptom; another symptom will then take its place. Injections of a local analgesic may be tried; they generally have a dramatic effect. Even as little as a few drops of 1% procaine may lead to fainting and hysterical manifestations.

One of us has attempted to treat the patients in groups by general ultra-violet irradiation followed by exercises; this method has had some success. Group psychotherapy of selected patients has now been started and has already given encouraging results. It is probable that an approach along these lines will prove to be the most profitable measure.

Summary

An investigation by a physician, a psychiatrist, and a psychologist of a group of patients suffering from a rheumatic disorder without signs of organic disease is described; a comparison with a control group is made.

In the former group the disorder was found to have certain special characters; in most cases the onset and course of the condition could be correlated with emotional disturbance. The condition seems to merit recognition as a clinical entity: it may be described by the term "psychogenic rheumatism."

We are indebted to Dr. T. A. Munro for his co-operation and advice; to Dr. K. W. N. Palmer, of the Department of Physical Medicine at Guy's, for his assistance with the control patients; and to Professor Aubrey Lewis and Dr. J. L. F. Maine for helpful suggestions.

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A CASE OF RHEUMATOID ARTHRITIS SIMULATING PYOGENIC INFECTION

BY

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The rarity of high fever in rheumatoid arthritis may be judged from the fact that Bannatyne (1896) on one occasion recorded a temperature of 105° F. (40.6° C.). This observation was later quoted by Garrod (1907) and by Jones (1909) as still unrivalled. Modern authors confine themselves to the statement that the temperature may rise to 102–103° F. (38.9–39.4° C.) and that a slight pyrexia may persist for several weeks.

The highest leucocytosis recorded in an uncomplicated case appears to be the figure of 15,800 mentioned by Erving (1903). Most modern writers omit all reference to the possible occurrence of a marked leucocytosis, and Gibson (1948) declares that "an increase of over 12,000 leucocytes per c.mm. is rare."

The following case is more reminiscent of the disease as seen in children, when fever and general symptoms may be present for weeks or months before definite articular manifestations make their appearance and fever and leucocytosis may be so marked as to simulate a pyogenic infection or leukaemia.

Case Report

The patient, a clerk aged 26, had previously been healthy save for a succession of sore throats.

He was first seen at Southmead Hospital on Jan. 2, 1948, when he gave a history of having had a sore throat nine weeks previously, accompanied by fever and pains in many joints, which had not become swollen. Fever with profuse sweating had persisted, and a temperature chart which he produced showed an evening rise varying between 101 and 103° F. (38.3 and 39.4° C.) over the past two and a half weeks.

On examination he was seen to be thin and pale. His temperature was 101.2° F. (38.4° C.) and the skin warm and moist. The pulse (90–120) was accelerated in proportion to the fever, and the B.P. was 130/80. The only joint affected was the left elbow, which showed a 30-degrees limitation of extension. There was no local heat, swelling, or tenderness, though muscle spasm was pronounced. Examination of all other systems was entirely negative, and no nodules were found. There was a soft systolic murmur at the apex.

Three days after admission an effusion developed in the left knee, and the metacarpo-phalangeal joint of the right thumb also became painful. A blood count revealed a leucocytosis of 30,800 (neutrophils 88%, lymphocytes 8%, metamyelocytes 4%); Hb, 72%, red cells, 3,790,000. Two blood cultures were sterile, and agglutinations to *Salmonella typhi, S. paratyphi* A and B, and *Brucella abortus* were negative. Radiographs of the chest and left elbow were normal. No abnormality was found in the urine, although a trace of albumin appeared in several subsequent specimens. No malaria parasites were found in the blood. The Wassermann reaction and Paul–Bunnell test were negative, and intravenous pyelography revealed no abnormality.

On the evening of Jan. 7 the patient had a temperature of 103.6° F. (39.8° C.) and appeared critically ill. It was considered justifiable to employ chemotherapy empirically. "Sulphamezathine" was accordingly given in full doses for a week, to a total of 50 g. By the time the drug was discontinued he appeared much improved: the effusion in the left knee had subsided, the temperature had been subnormal for five days, and the leucocyte count had fallen to 8,700.

During the following week the temperature started to rise again, until on Jan. 22 it reached 102° F. (38.9° C.). The leucocyte count reached 15,000 and the E.S.R. was 35 mm. in the first hour. Two further blood cultures were negative. A radiograph of the spine and long bones showed no evidence of osteomyelitis or other bony disease. An evening temperature of 102° F. was maintained for 72 hours, then gradually subsided to normal, where it remained for nine days. At the end of this time the patient felt well and did not complain of joint pains. The leucocyte count of 17,800 nevertheless revealed that all was not well.

The temperature started to mount on Feb. 6, and by Feb. 19 the patient was again seriously ill, with a leucocytosis of 24,000. Penicillin was begun in a dosage of 30,000 units three-hourly, and as there was no response within four days this drug was supplemented by sulphamezathine in full doses for six days. For three days the temperature fell to a lower level, but on the fourth day he had a rigor and it rose to 105.2° F. (40.7° C.). On the next two days there were further rigors, and temperatures of 104 and 105.2° F. (40 and 40.7° C.) were recorded. Forty-eight hours later the patient was once again afebrile.

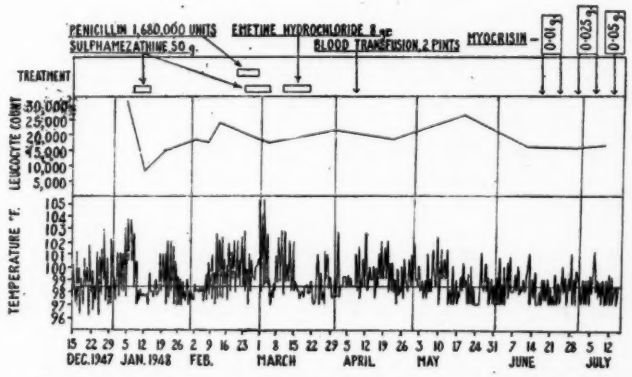
Further bouts of fever occurred during the next four months, and the temperature did not subside finally until the middle of July. During the first eighteen weeks in hospital the only constant physical sign was limitation of extension of the left elbow, with spasm and progressive wasting of the muscles above and below the affected joint. This and other joints were repeatedly *x*-rayed, with negative results until May 14, when periosteal changes were apparent around the neck of the left radius. Many joints were affected for short periods from an early stage in the course of the disease. At the end of February the left wrist and the proximal interphalangeal joints of the left hand became swollen and painful for a few days. Early in March there was pain on movement of the cervical spine, which persisted for about a fortnight. Later in the month both shoulders became painful for a week. In the middle of April both wrists were swollen and the proximal interphalangeal joints of the fingers were stiff for several days.

On May 17 more persistent changes appeared. The metacarpophalangeal joints of the left hand became swollen, red, and painful. At this time the leucocyte count rose to its second highest figure, 26,200. Ten days later the left wrist was inflamed and movement of the right wrist was found to be restricted. Thenceforward the typical deformity of the hands developed rapidly, and by the end of June radiological appearances characteristic of rheumatoid arthritis were apparent. None of the other joints, with the exception of the left elbow noted above, showed any radiological changes.

During the last week of February the patient complained of great soreness in the throat and pain on swallowing. The pharynx was injected, but no pathogens were isolated from the nose and throat swabs. Early in March definite hepatic tenderness became evident and there was an increase of urobilin in the urine. The van den Bergh reaction was negative, but the serum alkaline phosphatase was raised to 26.1 units. The serum albumin was 3.2 g. % and the serum globulin 4.1 g. %. In view of these observations, and bearing in mind that the patient had seen tropical service, amoebic hepatitis was considered. Emetine was tried empirically, 8 gr. (0.52 g.) of the hydrochloride being given over a period of ten days, but without benefit. In April one of the left axillary lymph glands became enlarged and was removed for biopsy. Non-specific inflammatory changes only were found.

Therapy and Progress

Sodium salicylate had been given for several weeks before admission and was administered again, without benefit, while the patient was in hospital. (For other therapeutic details, see Chart.) On July 29 he was transferred to the Royal National Hospital



Record of temperature, total leucocyte count, and treatment.

for Rheumatic Diseases, Bath, for further treatment. On admission his temperature was found to be 101.5° F. (38.6° C.) and for three weeks there was an evening rise of two or three degrees. He was given a transfusion of 2 pints (1.14 litres) of packed red cells. Other therapeutic measures included rest plasters for his hands at night, exercises in the pool, and mud-packs. Gold therapy was continued in the form of "aurocalcium," 20 mg. per week, rising later to 50 mg. At the end of November, when he was readmitted to Southmead Hospital for tonsillectomy, he looked remarkably well. His weight, which during April and May fell to 6 st. 12 lb. (42.6 kg.), had risen to 9 st. 9 lb. (60.5 kg.), and he had recovered a full head of hair, much of which had fallen out during his long illness. The only disability apparent was slight ulnar deviation of both wrists, with considerable limitation of movement in all directions. There was also periarticular thickening of the metacarpo-phalangeal and proximal interphalangeal joints of both hands, together with wasting of the intrinsic muscles. Radiographically, the carpus showed marked decalcification and narrowing of the joint spaces. All other joints (including the left elbow) showed a full range of painless movement.

Summary

A case of rheumatoid arthritis is described in which bouts of high fever occurred over a period of three months and of low-grade fever continued for a further four months before the diagnosis was evident. On two occasions a temperature of 105.2° F. (40.7° C.) was recorded after a rigor.

The highest leucocyte count was 30,800, recorded on admission; the next highest was 26,200 eighteen weeks later.

My thanks are due to Dr. G. D. Kersley for his help and for details of the patient's treatment and progress while under his care in the Royal National Hospital for Rheumatic Diseases, Bath; to Dr. D. H. Davies, under whose care the patient was admitted to Southmead Hospital, for advice and criticism; and to Dr. P. Phillips, the medical superintendent, for permission to publish.

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FIBROCYSTIC DISEASE OF THE PANCREAS*

BY

W. J. MATHESON, M.D., M.R.C.P., D.C.H.
(From the Paediatric Unit, St. Mary's Hospital, London)

[WITH PHOTOGRAVURE PLATE]

Although the American literature on this subject is now fairly extensive there have been few reports from this country. Doubtless many cases have gone unreported, but there is still a widespread belief that the disease is rare, and personal experience has shown that it is often misdiagnosed. The only large series described here is that of Bodian (1946), who reported 30 cases. Henderson (1945) recorded one case and MacGregor and Rhaney (1948) two cases in siblings. Previously, under the title of congenital steatorrhoea, Miller and Perkins (1920) and Cockayne (1933) each described one probable case of fibrocystic disease, and Clarke and Hadfield (1924) a proved case, for necropsy showed the pancreatic lesions which are now associated with this disease. It is unlikely that the case described by Garrod and Hurtley (1913) under the same title was one of fibrocystic disease, since Garrod (1920) subsequently reported that this patient was of normal development and activity at the age of 14 years.

Although the association of pancreatic disease and steatorrhoea had been known for over a century it was not until 1905 that Landsteiner described the pancreatic lesions now associated with fibrocystic disease. Passini (1919) first reported a case in which the association of the digestive disturbance and the pathological lesion of the pancreas was recognized. Parmelee (1935) described the cases of two patients with congenital steatorrhoea who died of bronchopneumonia, and in both of whom necropsy revealed cystic fibrosis in the pancreas; he had found in the literature only four cases with a similar history and post-mortem findings. Blackfan and Wolbach (1933) had, however, described six similar cases among 10 reported as cases of vitamin-A deficiency in infants, and Harper (1930) one case among two reported under the title of congenital steatorrhoea.

*Based on a thesis submitted to the University of Edinburgh for the degree of M.D., September, 1948.

Harper (1938) reported eight further cases under the same title, in five of which necropsy showed typical pancreatic lesions, and Blackfan and May (1938) described 35 cases with similar pancreatic lesions which in lesser degree they had observed in a further 200 cases; but they considered that it was not possible to determine accurately dependable diagnostic features in life in these cases. Fibrocystic disease of the pancreas was, however, becoming recognized with increasing frequency at necropsy; and, finally, Andersen (1938) showed that the disease was a definite and common clinical entity with a characteristic course and clinical history. She has since written extensively about the disease in all its aspects, and recently gave in this country a summary of her work (Andersen, 1949).

Case 1

This patient, a boy aged 10 (Fig. A), was first seen at the age of 8. Since birth his stools had been bulky, loose, pale, and offensive. Despite a good appetite and an adequate diet, he had never gained weight well. An irritative cough had been present since the age of 2 years, and had become worse and been accompanied by purulent sputum in the past nine months since an illness diagnosed as pertussis and bronchopneumonia. When 2 years old he was taken to another hospital, where coeliac disease was diagnosed. Dietary treatment for two years brought no improvement, and the diagnosis was altered to tabes mesenterica when his Mantoux reaction was found to be positive and calcified glands were seen in the abdomen at laparotomy performed on account of recurrent abdominal pain. He had had bronchopneumonia at 2 weeks and rectal prolapse at 6 months. His parents were not blood relations. One sibling aged 8 months is alive and well. The patient's weight was 44 lb. (19.96 kg.) (average, 57 lb.—25.85 kg.); height 48 in. (122 cm.) (average, 51 in.—130 cm.). His abdomen was prominent and his fingers were clubbed. Chest: percussion note hyperresonant; persistent crepitations in right lower and mid zones.

FIG. A.—Case 1 (on left) with normal boy of same age.

Investigations.—Duodenal juice contained no trypsin. Microscopical examinations of the stools revealed numerous fat globules and completely undigested muscle fibres; the fat content was 50%, of which 80% was split fat. Nitrogen excretion on a normal diet was 4.47 g. per day in faeces and 7.8 g. in urine. Blood counts showed no anaemia, but persistent polymorphonuclear leucocytosis. The predominant organism in the sputum was a penicillin-sensitive pneumococcus. Radiography of the chest showed emphysema with a generalized bronchitic appearance and parenchymal changes at the right base.

Progress and Treatment.—For 18 months he has been treated with a low-fat, high-protein, and high-carbohydrate diet and 30 g. of casein hydrolysate and 16 g. of pancreatin in the form of enteric-coated capsules and extra vitamins. He has gained only 4 lb. (1.81 kg.) in weight and 2½ in. (6.25 cm.) in height. At first his activity was greatly increased, but accesses of the pulmonary infection, although relieved by penicillin inhalations, have recently been occurring so often (about once a month) that his activity is now about the level maintained before specific therapy was started. Stools are now formed and not very offensive. On the same diet faecal nitrogen has been found to be diminished when pancreatin is given.

G. H. JENNINGS, J. L. HAMILTON-PATERSON, and F. O. MacCALLUM: FATAL CASE OF POLIOENCEPHALITIS DUE TO POLIOMYELITIS VIRUS

FIG. 1.—Normal anterior horn cell. H. & E. (× 1200.)

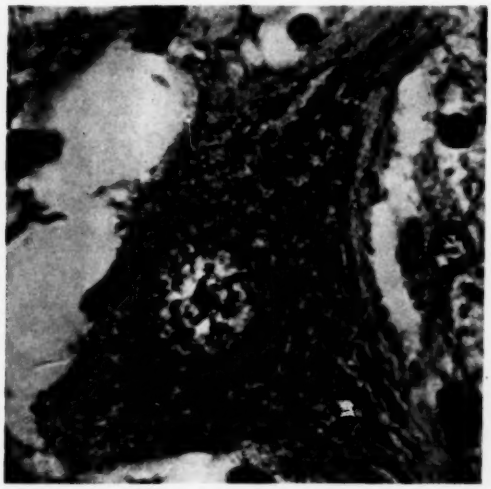

FIG. 2.—Anterior horn cell showing loss of nuclear membrane and nucleolus; partial granulation of tigroid substance. H. and E. (× 1200.)

FIG. 3.—Complete loss of nucleus and complete granulation of tigroid substance. H. and E. (× 1200.)

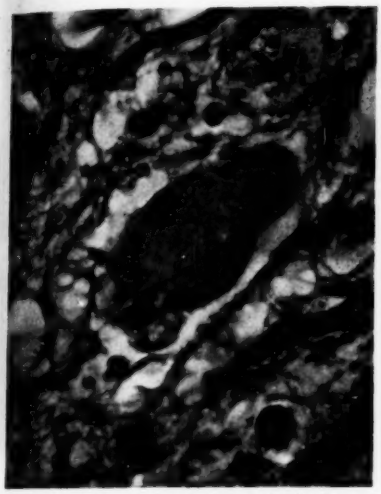

FIG. 4.—Complete chromatolysis, leaving a structureless eosinophil mass. H. and E. (× 1200.)

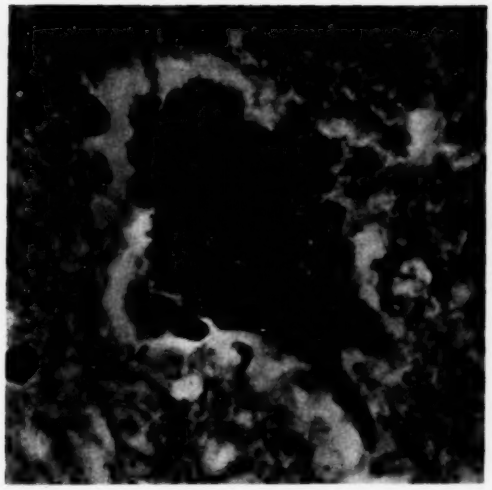

FIG. 5.—Commencing invasion of degenerated cell by histiocytes (monkey). H. and E. (× 1200.)

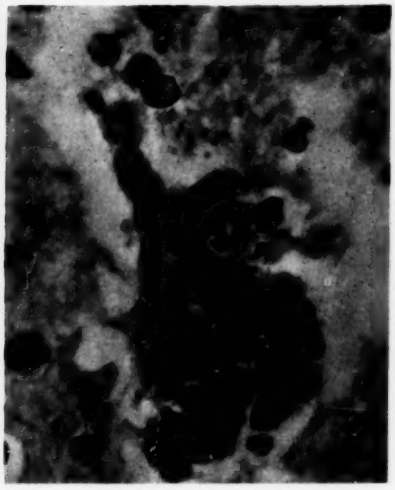

FIG. 6.—Degenerated neurone undergoing phagocytosis (monkey). H. and E. (× 1200.)

BERNARD SCHLESINGER: RHEUMATOID ARTHRITIS IN THE YOUNG

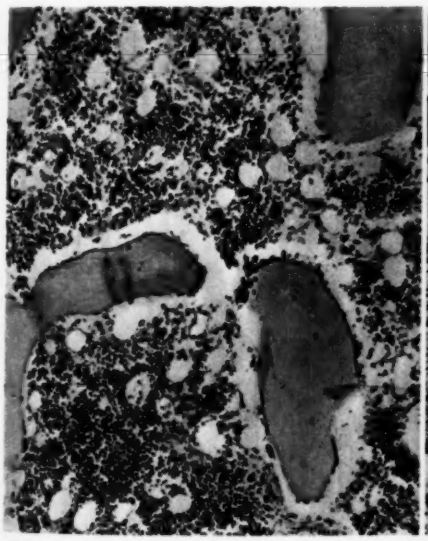

FIG. 1.—Normal bone-marrow section at necropsy.

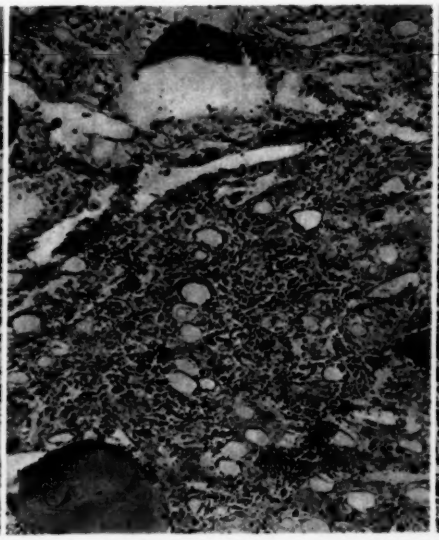

FIG. 2.—Section of bone marrow from femur of Case 1. Hypocellular, with areas of necrobiosis.

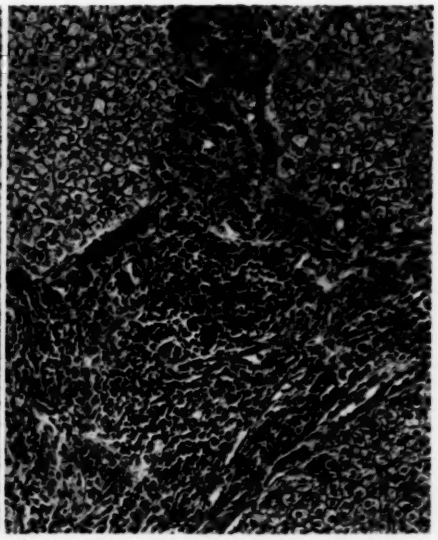

FIG. 3.—Section of liver of Case 11 showing perilobular fibrosis and infiltration of inflammatory cells.

W. J. MATHESON: FIBROCYSTIC DISEASE OF THE PANCREAS

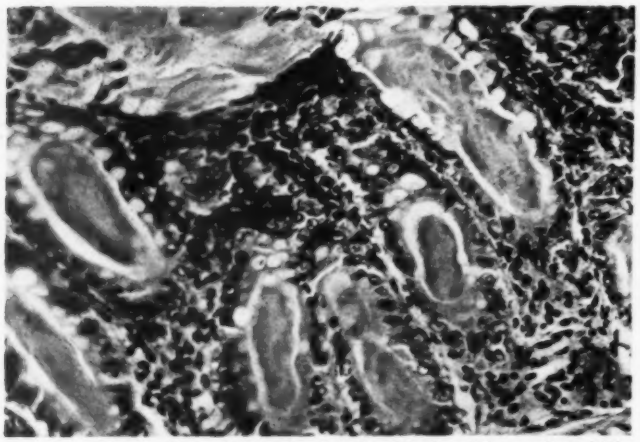

FIG. 1.—Appendix. Dilated glands filled with eosinophilic material, in places continuous with similar material filling the lumen. (× 190.)

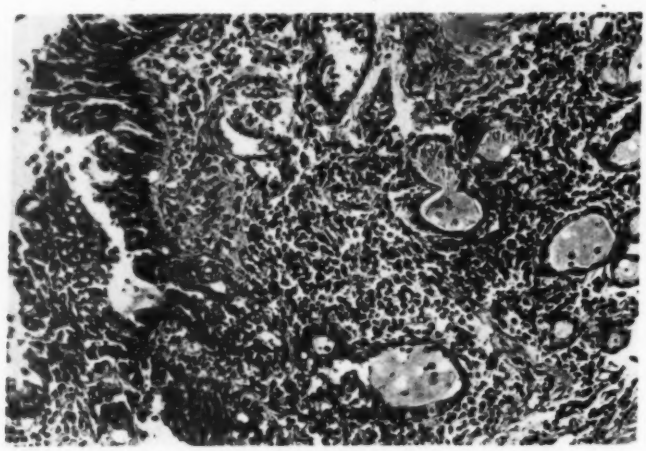

FIG. 2.—Bronchus showing dilated glands in wall. (× 105.)

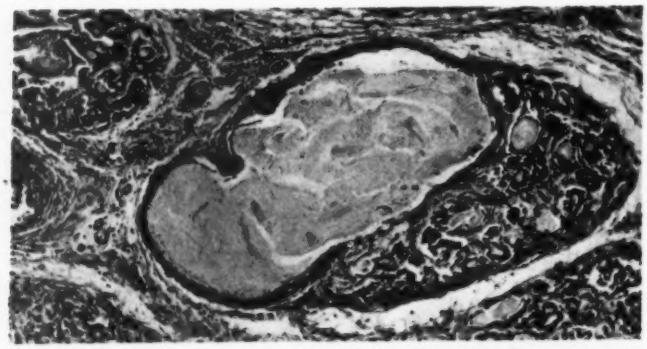

FIG. 3.—Pancreas. Grossly dilated duct lined by flattened cells and filled with eosinophilic material, as are the smaller ducts. (× 60.)

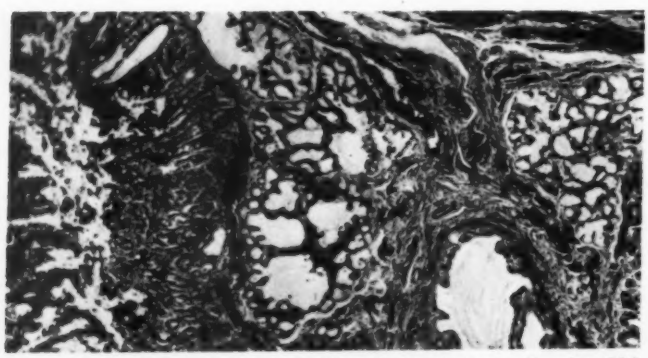

FIG. 4.—Duodenum showing dilated glands in wall. (× 90.)

S. N. DE: STREPTOMYCIN IN DEVELOPMENT OF HYDROCEPHALUS IN TUBERCULOUS MENINGITIS

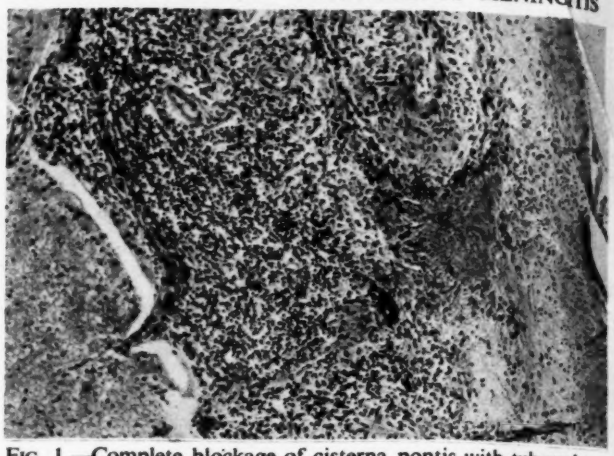

FIG. 1.—Complete blockage of cisterna pontis with tuberculous granulation tissue. Hydrocephalus followed meningitis not treated with streptomycin. H. and E. (× 65.)

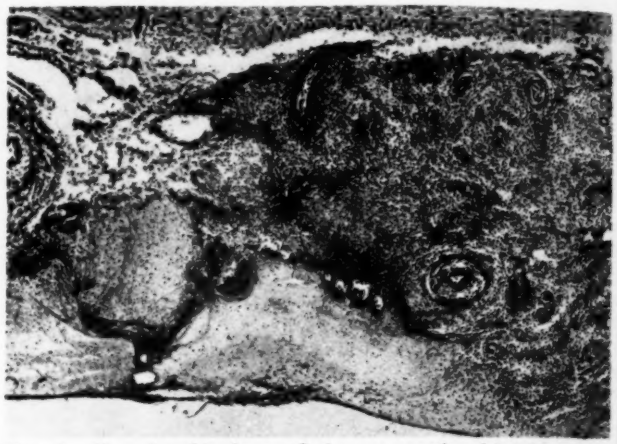

FIG. 2.—Complete blockage of cisterna pontis with tuberculous granulation tissue. Hydrocephalus followed meningitis treated with streptomycin. H. and E. (× 25.)

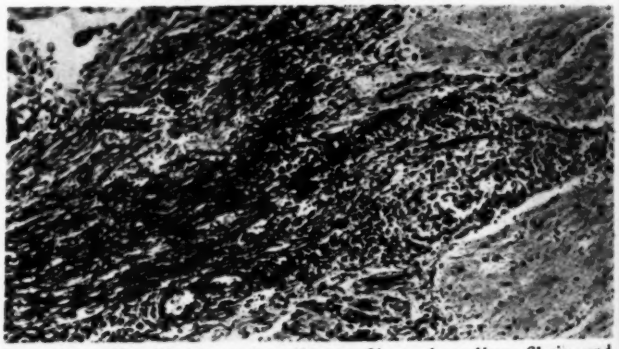

FIG. 3.—Fibroblasts and collagen fibres invading fibrin and tubercles within meningeal exudate. Hydrocephalus followed meningitis treated with streptomycin. H. and E. (× 65.)

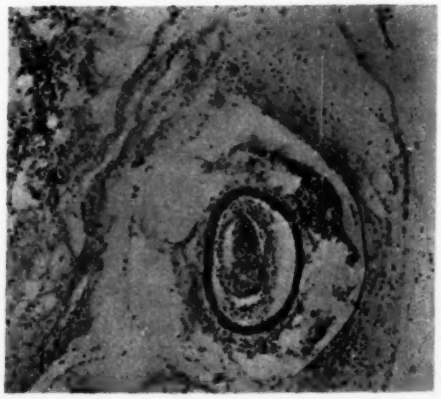

FIG. 4.—Patent subarachnoid channels within cisterna pontis. Tuberculous meningitis treated with streptomycin. No hydrocephalus. H. and E. (× 45.)

Illustrations printed in Collogravure by HARRISON & SONS, LTD., Printers to His Majesty The King, London, Hayes (Middx.) and High Wycombe.

Case 2

A boy aged 2 years was admitted to hospital on Sept. 13, 1948. Since the age of 2 months he had wheezed continually; a diagnosis of asthma was made and never altered. One month before admission he began to cough and lost weight and energy. Rectal prolapse had occurred repeatedly in the past two months. Despite a good appetite, he had never thrived, his weight at 8 months being 15½ lb. (7 kg.) and at 1 year 18½ lb. (8.4 kg.) (birth weight was 7½ lb.—3.4 kg.). From the age of 6 months or so his stools had been frequent and unformed, bulky, and very offensive. He was the third child. The eldest died of acute bronchitis at 11 weeks. The second, aged 5 years, is alive and well. A paternal aunt, who had never thrived, died at 16 days.

On examination his temperature was normal, pulse 120, and respirations 32; he was dyspnoeic, with frequent paroxysmal coughing. His weight was 21 lb. (9.5 kg.) (average, 27 lb.—12.2 kg.); height, 30 in. (76 cm.) (average, 33 in.—83.5 cm.). Chest: percussion note hyperresonant; rhonchi and crepitations in all areas. The liver was palpable two fingerbreadths below the costal margin in the nipple line.

Investigations.—Numerous fat globules and undigested muscle fibres were found in the stools; the fat content was 30%. A laryngeal swab revealed *Staphylococcus aureus* sensitive to streptomycin only. Duodenal intubation was unsuccessful. Blood counts showed a progressive fall in haemoglobin to 75% on Oct. 28; there was a persistent polymorphonuclear leucocytosis up to 40,000 per c.mm. Radiographs of the chest showed increasing spread and confluence of bronchopneumonia in all areas of the lung fields.

Progress and Treatment.—Wheezing disappeared after two weeks, but despite the use of streptomycin, given by inhalation and intramuscularly, the cough became more severe, crepitations persisted in the lungs, and a swinging temperature soon developed and persisted. Cyanosis was present in the fortnight before death on Nov. 19. His appetite had been good for six weeks, and on a low-fat diet with extra protein and 26 g. of casein hydrolysate and 8 g. of pancreatin (in the form of enteric-coated granules) daily his weight had risen to 22 lb. (9.98 kg.) but then fell progressively. His stools had continually been frequent, unformed, and very offensive. The liver had remained enlarged.

Post-mortem Report.—There was much mucopurulent material in the air passages, and in the lungs extensive bronchopneumonia with bronchiolectatic abscesses. The pancreas appeared normal macroscopically. The liver was "nutmeg," and the appendix was thickened. Microscopically, the pancreatic ducts were considerably dilated and filled with eosinophilic material, and there was much surrounding fibrosis; the lumen of the appendix was filled with eosinophilic material continuous with that in the dilated glands of the wall (Plate, Fig. 1). Similar lesions were seen in the mucous glands of the trachea and bronchi (Fig. 2), the salivary glands, the duodenal wall, and the periurethral glands.

Case 3

A girl aged 2 years was first seen in January, 1947, at the age of 21 months, with rectal prolapse, which was attributed to inadequate intake of vitamin D. Her weight was then 24 lb. (10.88 kg.). When next seen, a month later, she had developed a slight cough and had lost weight despite a good appetite. She was admitted early in May, 1947, for correction of the prolapse, but was discharged five days later as she was thought to have pertussis. At home the cough became more severe, the weight-loss was accelerated, and she was readmitted at the end of May. Even in infancy her gain in weight had never been satisfactory, although her appetite was so good that she was fed three-hourly with amounts prescribed for four-hourly feeds. Her stools had always been bulky, pale, and very offensive.

She was the fourth child of healthy unrelated parents. Her three siblings had all died of bronchopneumonia before the age of 6 months; none had thrived, and all had had very offensive stools. The maternal grandmother had lost five out of eight children in infancy from unknown causes. The paternal grandmother had lost six of 12 children in infancy—four at birth and two of pneumonia during the first year.

On May 22 her temperature was 101.6° F. (38.7° C.), respirations 64, pulse 128, weight 18¼ lb (8.28 kg.) (average, 24 lb.—10.88 kg.). She was wasted and dyspnoeic. Chest: Fine crepitations in all areas. The abdomen was protuberant. The liver was enlarged to the umbilicus. The spleen was not palpable.

Investigations.—The faecal fat amounted to 49%, 86% of it being split fat. Blood counts showed a progressive anaemia. Three attempts at duodenal intubation were unsuccessful. Mantoux, negative at 1 in 1,000. Serum proteins (June 25) were 7 g. %—albumin 2.8, globulin 4.2. Chest radiographs showed increasing bronchopneumonic changes.

Progress and Treatment.—Sulphamezathine and penicillin failed to control the respiratory infection. Cyanosis was only temporarily relieved by oxygen. The stools were frequent, unformed, and very offensive; appetite was never good, and weight fell progressively. Pancreatin given in the last three weeks had no appreciable effect. Peripheral oedema, unrelieved by blood transfusion and intravenous "casydrol," appeared four weeks before death, and petechiae terminally.

Post-mortem Report (July 22).—Much mucopus in the air passages. In the lungs marginal emphysema was present, with extensive bronchopneumonia and numerous bronchiectatic abscesses in practically all areas. The pancreas on section showed dilated ducts. The liver was large and pale. Microscopical examination showed gross dilatation of pancreatic ducts filled with eosinophilic material, much surrounding fibrosis, and almost complete disappearance of acinous tissue. There was bronchopneumonia with, in places, destruction of bronchial walls and dilated bronchial glands. The liver showed gross fatty degeneration.

Case 4

A girl aged 4 months was transferred on July 16, 1948, from another hospital, where she had been treated for six weeks for bronchopneumonia without improvement. Despite a good appetite she had never thrived, and her stools had always been frequent and offensive. At 3 months she developed pneumonia, which did not respond to treatment with sulphonamides and penicillin. She had three siblings alive and well. A maternal uncle, who had never thrived, died at 10 years of "consumption of the bowels."

On examination on July 16 the temperature was 99° F. (37.2° C.), respirations 48, weight 8 lb. 15 oz. (4 kg.) (expected weight, 12½ lb.—5.67 kg.). She was wasted; the abdomen was prominent. There was dyspnoea with frequent cough. Chest: percussion note impaired at left base; fine crepitations audible over the entire left chest.

Investigations.—Duodenal juice contained no trypsin. Faecal fat: (*a*) on skimmed milk 40%, (*b*) on half-cream milk with pancreatin 21% (96% split). A laryngeal swab contained predominantly a penicillin-sensitive *Staph. aureus.* Hb was 65%. Radiography of the chest showed left basal consolidation.

Progress and Treatment.—Penicillin was continued, but the respiratory trouble spread. Appetite was poor. Stools were always frequent, bulky, and very offensive. She died on Aug. 4.

Post-mortem Report.—The immediate cause of death was bronchopneumonia. The pancreas was smaller than normal, was nodular, and presented an increased resistance to section. Microscopically, typical changes were seen in the pancreas and salivary glands.

Case 5

A boy aged 4 months was admitted to hospital on Aug. 3, 1948. His appetite had always been good, but weight-gain had never been satisfactory (his best weight was 8 lb. (3.63 kg.) at three months). Stools had always been frequent but not noticeably offensive. A frequent dry cough had been present since birth.

On examination the temperature and respirations were normal and the weight 7 lb. 11 oz. (3.48 kg.) (expected weight,

12¼ lb.—5.55 kg.). Mediastinal shift to right with hyperresonant percussion note over left chest.

Investigations.—Three attempts at duodenal intubation failed. Faecal fat (on full-cream milk), 72% (50% split). Laryngeal swab revealed *Staph. aureus.* Blood counts showed a progressive anaemia.

Progress and Treatment.—Ten days after admission he developed a pulmonary infection which did not respond to penicillin. Appetite was very good, and on a skimmed-milk diet with pancreatin and hydrolysates weight increased slowly to 8 lb. 10 oz. (3.91 kg.). Stools were always bulky, frequent, and very offensive.

Post-mortem Report (Sept. 21).—The immediate cause of death was bronchopneumonia. The pancreas was normal externally, but dilated ducts were seen on section. Microscopically, typical changes were seen in the pancreas, salivary glands, and bronchial and intestinal walls.

Case 6

A girl aged 5 months, an only child, was admitted to hospital on Nov. 1, 1947, with right basal pneumonia. She had had a cough since birth, but weight-gain had been satisfactory, and she had reached 12¼ lb. (5.55 kg.) (expected weight, 12 lb. —5.44 kg.) when pneumonia developed two weeks before admission. Chemotherapy and antibiotics failed to control the respiratory infection, and although she ate well her weight fell. Stools were first noticed to be offensive two weeks after admission; the fat content was 40% (on half-cream milk feeds).

Post-mortem Report (Dec. 17).—The immediate cause of death was bronchopneumonia. The pancreas was smaller and more nodular than normal. On the surface of the liver were small nodules up to 2 mm. in diameter. Microscopically the pancreas showed the typical changes in an advanced stage (Fig. 3). In the liver the nodules were seen macroscopically to consist of cirrhotic areas in which were numerous bile ducts filled with eosinophilic material. Similar lesions to those in the pancreas were found in the salivary glands and in the duodenal wall (Fig. 4).

Case 7

A girl aged 16 months was transferred on July 30, 1948, from another hospital, where she had been treated for two months for coeliac disease without improvement. Weight-gain was never satisfactory, and from the age of 4 months her stools were bulky and offensive. This abnormality became more noticeable at 1 year. She had had bronchitis at 8 months.

On July 30 her weight was 15¼ lb. (6.92 kg.) (average, 22 lb. —9.98 kg.). She had a coeliac appearance. Her chest was normal. Duodenal juice showed tryptic activity in undiluted juice but not in a dilution of 1 in 10. Faecal fat: (*a*) on a normal diet, 60% (75% split; (*b*) on a low-fat diet with pancreatin, 15%. With specific dietary treatment she gained 3 lb. (1.36 kg.) in three weeks. Her stools were always unformed and offensive.

Discussion

The seven cases described were seen between June, 1947, and September, 1948. To ascertain the hospital incidence it is necessary to exclude Cases 4 and 5, which were transferred from other hospitals because of our known interest in fibrocystic disease; the remaining five cases occurred among 240 medical admissions to the children's ward—an incidence of 2%. Andersen (1938) found evidence of the disease in 3% of all necropsies on infants, and Bodian (1946) in 3.4% of 500 necropsies on children. The disease therefore is by no means uncommon. The failure to appreciate this prior to 1938 is undoubtedly due to the fact that bronchopneumonia, the usual cause of death, was taken to be an adequate cause and consequently the pancreas was not sectioned.

The evidence points to the disease being inherited (Andersen, 1949). In this series no other proved case has occurred in an affected family, but there is presumptive evidence of the disease in the family history of Case 3 and suggestive evidence in Case 2. The inheritance may be explained by incomplete dominance: Fisher (quoted by Cockayne, 1947) has shown that many dominant defects are gradually becoming recessive, this being brought about by the selection of genes which prevent a dominant condition becoming manifest when in the heterozygous state; moreover, a defect may behave in one family as a recessive and in another as a dominant.

The pancreatic lesions were formerly sometimes ascribed to dehydration, but Farber (1942) disproved this. Blackfan and Wolbach (1933) first advanced the hypothesis that they were caused by inspissation of viscid secretion in the ducts; this was confirmed by Farber (1942), who reproduced the lesions in kittens by vagal stimulation. It remains to be seen if similar experiments will reproduce the lesions occurring in other organs also, and the cause of the abnormal secretion has yet to be determined.

Farber (1944) reported the finding of dilated glandular structures in both the respiratory and the intestinal tracts; he concluded that the disease affected all mucus-secreting glands, the lesions being caused by inspissation of viscid secretion, and that it was the loss of normal mucus from the bronchial tree that was responsible for the susceptibility to pulmonary infections. Bodian (1946) also has stressed that this is a pluriglandular disease, as have Continental workers (Glanzmann, 1946; Riniker, 1946). Andersen (1949), however, while accepting Blackfan and Wolbach's explanation for the pancreatic lesions, considers that the pulmonary infection is conditioned by dietary deficiency, and that the lesions observed in the bronchial walls are secondary to infection. But this explanation does not suffice for the similar lesions found in other organs in which there is no infection; moreover, dogs in whom the pancreatic duct had been tied did not die from pulmonary infections (Ivy, 1936). The pathological findings in this series have again confirmed the pluriglandular nature of this disease, and there seems little doubt that Farber's hypothesis is correct.

Earlier observers (Parmelee, 1935; Harper, 1938) considered that a high percentage of neutral fat in the stools indicated pancreatic disease. Andersen (1945a), however, has shown that faecal fat can be split in the colon and outside the body by bacterial action, and Fourman *et al.* (1948) consider that increased nitrogen excretion in the faeces is the most reliable test of pancreatic steatorrhoea. Fat analyses of three-day collections of stools, as carried out in these cases, will usually detect steatorrhoea (Fourman *et al.*, 1948), but the determination of split and unsplit fat is of no diagnostic value (May, 1947).

Andersen (1938) showed that cases fell into three main groups: (1) cases of meconium ileus which present within a few days of birth with intestinal obstruction; (2) cases which present in the first six months of life with respiratory infections—nutritional difficulty is also present (this is the largest group); (3) cases which present later with nutritional difficulty as instances of the coeliac syndrome or with rectal prolapse or with chronic respiratory infection. The typical history is of a poor gain in weight despite an adequate diet and an excellent appetite, dating from early infancy. The stools are frequent, unformed, bulky, and very offensive; these abnormalities are more often noticed by mothers who have previously borne normal children, and usually date from early infancy. Sooner or later respiratory infection supervenes and dominates the picture.

Although it is generally agreed (May, 1947; Andersen, 1949) that estimation of trypsin in the duodenal juice is necessary to establish the diagnosis, nevertheless it is

desirable to make a diagnosis on clinical evidence whenever possible, and experience has indicated that this can be done. In all the present cases the diagnosis was made clinically and was never disproved by subsequent examination of the duodenal juice or by necropsy. The clinical picture is highly characteristic, but it must be emphasized that an accurate history is essential, and close questioning may be required to obtain this, as noted by Miller and Perkins (1920).

The disease should be suspected in all cases of chronic nutritional difficulty and chronic respiratory infection in children, and specific questions asked about appetite, weight-gain, the character of the stools in regard to number, size, and smell, the exact date of onset of any abnormality noted, and the family history, especially of infantile deaths. In a number of cases the abnormality of the stools, while present from early infancy, becomes more obvious when solids are added to the diet or even later, and this overshadows the history. Cough may be present without signs of infection, and is then presumably due to efforts to expel viscid mucus from the bronchial tree. The clinician should himself examine the stools, for their offensive smell is characteristic. The typical history, associated with the clinical findings, enables a firm diagnosis to be made.

Sheldon (1948) considers that it is usually possible to distinguish coeliac from fibrocystic disease on clinical grounds alone. In typical cases, therefore, duodenal intubation is superfluous and should be reserved for cases in which the picture is not characteristic—e.g., Case 6. It is important to remember that in some children any chronic infection may produce steatorrhoea and that the association of bronchiectasis and steatorrhoea does not justify a diagnosis of fibrocystic disease without supporting evidence.

It is not only desirable but sometimes necessary to make a clinical diagnosis, since duodenal intubation has often been unsuccessful, and repeated attempts do not improve the condition of children already wasted. Moreover, the absence of trypsin from the duodenal juice is not pathognomonic of fibrocystic disease. Dr. G. M. Bull, to whom I am indebted for these particulars, has under his care two adults—brother and sister, aged 18 and 22 years respectively—who have suffered from steatorrhoea since early infancy. Recently duodenal intubation has shown a complete absence of trypsin in both, but there is no pulmonary infection. These patients are not suffering from fibrocystic disease as regarded generally. Their cases are, however, similar to the case reported by Garrod and Hurtley (1913). Either the underlying lesion may be a congenital stenosis of the main pancreatic duct or these cases may be analogous to that reported by Farber (1944) in which lesions typical of fibrocystic disease were found only in the lungs and liver; vice versa, there may be cases in which the pancreas is affected and not the lungs.

Treatment.—The diet used has been that recommended by Andersen (1949), but pancreatin has been more extensively employed than casein hydrolysates, since the latter are unpalatable and older children are loath to take them. It is unfortunate that hydrolysates are so unpleasant, since the evidence indicates that they are of more value than pancreatin (West *et al.*, 1946).

Results of Treatment and Prognosis.—Of the seven patients, five have died of bronchopneumonia and one of the survivors has extensive bronchiectasis. The prognosis therefore depends on the respiratory infection, and the failure of appropriate chemotherapeutic and antibiotic agents to control it has been most disappointing. Specific dietary treatment also has proved of limited value. Pancreatin does, however, seem to be of value in increasing the utilization of food, and this effect is especially noticeable in infants.

The number of cases reported here is small, but no evidence has been found to support Andersen's (1949) contention that appropriate chemotherapy, given early in the course of the respiratory infection, combined with specific dietary treatment, may result in the control of the infection. Personal experience of cases diagnosed before the onset of respiratory infections is limited, but Andersen's (1945b) evidence that most, when given specific therapy, do not contract chronic respiratory infections and develop normally is not fully convincing. In assessing the results of any therapeutic regime in this disease it is important to remember that the oldest proved case in the literature, aged 14½ years (Parmelee, 1935), was treated by a low-fat and high-protein diet only. Moreover, Cases 1 and 7 in this series showed that pulmonary infections in infancy do not invariably become chronic even without treatment. Also there are a few cases which, although they lack trypsin in their duodenal juice, do not develop pulmonary infections at all.

Since in the great majority of cases of bronchopneumonia in children a good response is obtained, the failure of chemotherapy to control the pulmonary infections in these cases suggests that unusual factors are involved. One unusual factor is the abnormality of the mucous glands of the air passages, and the treatment so far possible cannot be expected to influence this. The rational treatment would be to liquefy the mucus in the bronchial tree by some substance (e.g., hyaluronidase) aimed at restoring normal conditions in the respiratory system.

Summary

Seven cases of fibrocystic disease of the pancreas are described which occurred among 2% of routine admissions to a children's ward.

Inheritance by incomplete dominance is postulated.

The pathological findings have again confirmed that this disease affects mucus-secreting glands throughout the body.

The diagnosis can usually be made clinically provided an accurate history is obtained.

The results of treatment have been disappointing, since five of the patients have died of bronchopneumonia and one of the survivors has extensive bronchiectasis.

It is suggested that measures directed at liquefying the mucus in the bronchial tree might lead to more successful results than those generally at present obtained.

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FATAL CASE OF POLIO-ENCEPHALITIS DUE TO POLIOMYELITIS VIRUS

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[WITH PHOTOGRAVURE PLATE]

The case of fatal polio-encephalitis described in detail below is reported because of its interesting clinical picture and because of the striking proof of the diagnosis by intracerebral inoculation in a monkey. The similarity of the lesions in the cord of the human victim to those in the monkey inoculated with a filtered saline extract from the child's brain and cord can easily be seen by reference to the illustrations.

Case Report

A well-developed child of 3½ years was admitted to Edgware General Hospital on Aug. 3, 1948. He had had pertussis at 1½ years, but had otherwise been well until he began to attend a nursery school one month before his admission. He was not happy at the school, slept irregularly at night, and was sleepy in the mornings. Three weeks before admission he had an illness of influenzal type lasting one day, during which he vomited and was febrile and drowsy. On July 31 he was said to have fallen from a swing, though apparently with no untoward effect. However, the next day he became drowsy and slept through much of it, would not eat, complained of abdominal pains, and vomited twice. During the night he awoke three times and complained of headache and pains in the back of the neck and in the legs. On Aug. 2 he still complained of the pains, and some weakness of the left side of the face developed, the face being drawn to the right. Although he was febrile and eager for fluids, he appeared to swallow with difficulty. He talked normally throughout the day. By evening he was generally stiff and showed a tendency to restlessness and twitching of the limbs. The bowels were constipated from that day to the end, and he again vomited once. On the day of admission he was not easy to rouse and would not speak; the restlessness and twitchings continued.

On admission he was very drowsy, toxic, and cyanosed. His temperature was 102° F. (38.9° C.), pulse 88, respiration 56. His tongue was moist but furred. The throat was not inflamed, but the tonsillar glands were palpable. There was pronounced stiffness of the neck and spine. Kernig's sign was not present and the legs were not stiff. His pupils were equal and reacted to light, though sluggish. The disks were normal. He had slight weakness of the left side of the face. No other apparent abnormality in cranial nerves was observed and no weakness was noted in the limbs. Tendon-jerks were absent in the left lower limb but brisk on the right side. Plantar response was extensor on the left side and indefinite on the right. No abnormality was noted in other systems.

Lumbar puncture showed clear fluid under normal pressure; total protein, 80 mg. per 100 ml.; Pandy +; white blood cells, 55 per c.mm (degenerate polymorphs 5%, lymphocytes 20%, large endothelials 75%); chlorides, 710 mg. per 100 ml.; no organisms seen; sterile on culture.

Overnight his condition had deteriorated rapidly, and when seen by one of us (G. H. J.) next morning (Aug. 4) he was almost completely unconscious. He was still toxic and cyanosed; respirations were stertorous, but the rate was only 22. His temperature was 98° F. (36.7° C.) and pulse 80. A tendency to show slight general convulsions was noted. The disks were normal, as were the pupils, apart from the persistent sluggish reaction to light. Neck stiffness was slight and Kernig's sign was negative. The face was now almost expressionless as a result of general weakness. The left side of the body, though still showing voluntary movement, appeared definitely weaker and more flaccid than the right. The tendon-jerks, though sluggish, were all elicited except the right ankle-jerk. The abdominal reflexes were all present. The left plantar response was still definitely extensor, but that on the right remained indefinite.

Lumbar puncture showed C.S.F. under very low pressure; no block; opalescent fluid; fibrin clot formed after 15 minutes; white blood cells, 195 per c.mm. (lymphocytes 86%, polymorphs 14%); red blood cells, 465 per c.mm.; normal sugar content; sterile on culture. A blood count showed: Hb, 90%; red cells, 5,090,000; white cells, 4,700 (polymorphs 68%, lymphocytes 28%, monocytes 4%.

A diagnosis of a neurotropic virus infection was made, and since the prodromal illness three weeks before admission was within the reported maximal incubation period of 35 days for the poliomyelitis virus it was considered that the case was possibly one of polio-encephalitis. The child's condition continued to deteriorate; the temperature became subnormal, the pulse rate increased to 120–136, and the respiration rate remained at 24–26. He died early on the following morning (Aug. 5) after many hours of complete unconsciousness.

Post-mortem Examination

This was performed five hours after death. The body was that of a normally developed male child.

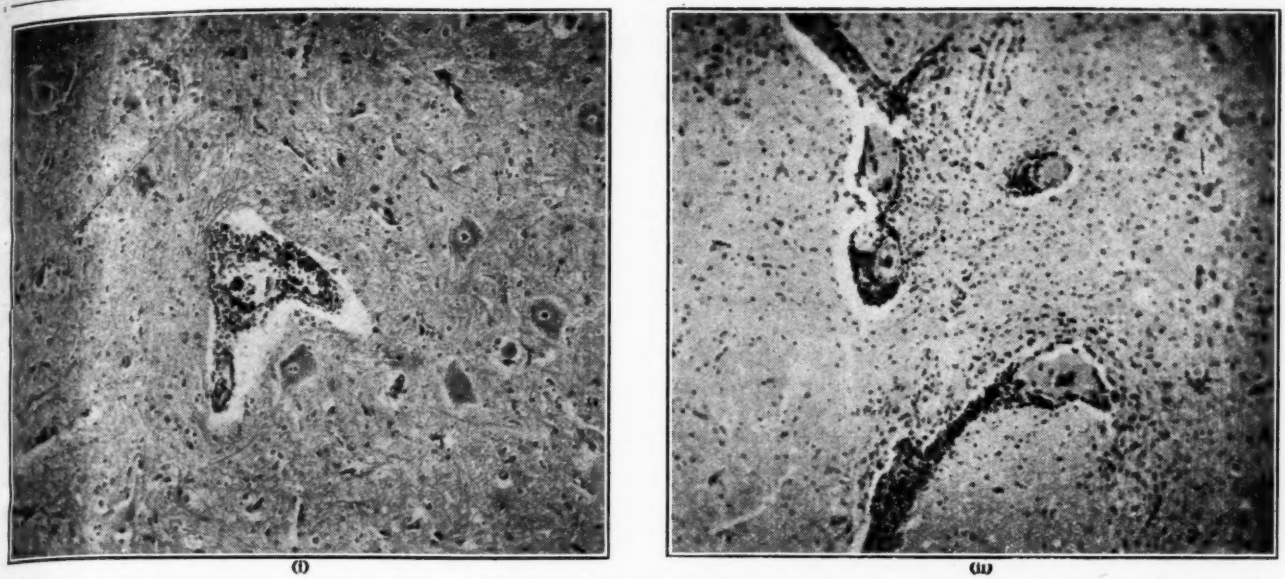
Brain (1,350 g.).—Meninges normal. Surface of brain intensely congested. On section the cortex of the hemispheres was plum-coloured, but showed no petechiae; white matter normal except for a few tiny petechial haemorrhages in the occipital lobes. The cerebellum, mid-brain, pons, and medulla were macroscopically normal.

Spinal Cord.—Macroscopically normal.

Skull.—Normal except for a small quantity of yellow glairy fluid in the right mastoid cavity. Films of this fluid showed occasional pus cells, and on culture a few colonies of *Bacterium coli* and *Streptococcus faecalis* were grown.

Comparison of Histological Lesions of Central Nervous System in the Case described and in the Inoculated Monkey

	Human	Monkey
Motor cortex, left	Congestion only	Early perivascular cuffing
„ „ right		Nil
Sensory cortex, left	—	Nil
„ „ right	—	Nil
Olfactory bulb ..	—	Nil
Basal nuclei	—	Nil
Brain stem	Few small areas of perivascular cuffing and very early neuronal changes	Perivascular cuffing and early neuronal degeneration
Cervical cord ..	Marked perivascular cuffing. Slight cellular infiltration of anterior horns. Early neuronal degeneration	Marked perivascular cuffing. Cellular infiltration of grey matter, most marked in anterior horns. All stages of neuronal degeneration, including neuronophagia by histiocytic cells. The changes increase in severity, progressing from cervical to lumbar region
Thoracic cord ..	—	
Lumbar cord ..	Marked perivascular cuffing. Marked cellular infiltration of anterior horns, particularly on left side. Advanced neuronal degeneration	



(i) (ii)

FIG. A.—Perivascular cuffing in poliomyelitis. (i) Section of lumbar cord (human). H. and E. (× 100). (ii) Section of lumbar cord (monkey). H. and E. (× 100).

The remaining organs were normal macroscopically and microscopically except for some fatty degeneration in the liver cells.

Various pieces of the nervous system were fixed in formol-saline for histological examination. Sections were stained with haematoxylin and eosin, Loyez's haematoxylin, and Mallory's phosphotungstic acid. The detailed changes noted in these tissues are shown in the accompanying Table and are compared with those of an inoculated rhesus monkey (see below) which had been similarly fixed and stained.

Animal Inoculation (Dr. F. O. MacCallum)

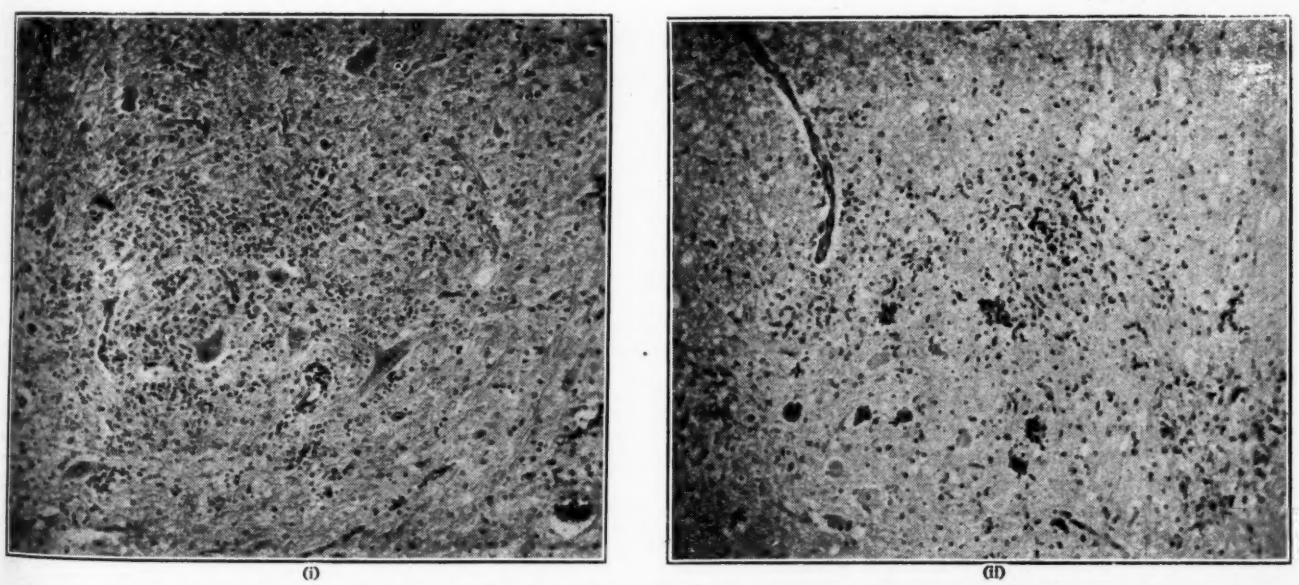
The diagnosis of poliomyelitis infection in this case was confirmed by animal inoculation. A suspension of pieces of cerebrum, mid-brain, and pons was prepared within a few hours of necropsy and inoculated intracerebrally and intranasally under ether anaesthesia into one monkey of the species *Cercopithecus aethiops sabaeus*. The monkey developed typical signs of poliomyelitis 13 days later and was killed with chloroform. Histological examination of the central nervous system revealed typical lesions of poliomyelitis.

A suspension of cord from this monkey was inoculated intracerebrally into a rhesus monkey, which also developed typical poliomyelitis eight days later; the microscopical lesions are well shown in Figs. A ii and B ii and in the Plate (Figs. 5 and 6). The original C.N.S. tissues of the patient were also inoculated intracerebrally into mice and guinea-pigs, with negative results.

Histological Findings

The histological changes in both the human and the monkey sections were basically the same and varied only in degree. The perivascular cuffing, neuronal degeneration, and cellular infiltration characteristic of poliomyelitis were all found in the spinal cord and brain-stem.

The neuronal degeneration (Plate, Figs. 1–6) showed itself as a loss of the nuclear membrane and of the oxyphil nucleolus (Fig. 2). Next the tigroid substance became granular or showed condensation at the side of the cell (Fig. 3). Eventually the nucleus and the tigroid substance completely disappeared, leaving a structureless eosinophil mass (Fig. 4). This was invaded by histiocytes (neuronophagia), which eventually removed it completely (Figs. 5 and 6). The cellular infiltration



(i) (ii)

FIG. B.—Cellular infiltration of spinal cord in poliomyelitis. (i) Anterior horn of lumbar cord (human). H. and E. (×100). (ii) Anterior horn of lumbar cord (monkey). H. and E. (× 100).

of the grey matter (Figs. B i and B ii) showed the same cells as in the perivascular cuffs. No demyelination of the white matter was seen.

The perivascular cuffing (Figs. A i and A ii) consisted of an infiltration of the perivascular space with histiocytic cells and lymphocytes; very few polymorphonuclear cells were seen.

Discussion

This case of polio-encephalitis had several features of interest apart from the confirmation of the diagnosis by animal inoculation. First among them comes the encephalitic nature of the signs, the chief of these being coma and evidence of brain-stem involvement. Kelleher (1947) stated that in his experience 30% of the poliomyelitis cases seen in the first part of the 1947 epidemic were of the bulbar type; of these a few were drowsy, and one became comatose and died after being unconscious for less than 24 hours. This last case had pronounced cranial-nerve disturbances together with mid-brain and cerebellar involvement. Brewis and Neubauer (1948) described a probable case of polio-encephalitis which recovered after showing coma, fits, cerebellar signs, and spastic weakness. Laurent (1947) recounted two similar examples of encephalitis, one of which made a good recovery; the other died 16 hours after admission. Necropsy in the latter case showed only oedema in the cerebral cortex, but small areas of perivascular "cuffing" with mononuclear cells were seen throughout the brain-stem (bulb, pons, mid-brain, and basal ganglia).

These findings correspond to some extent with those in our case, in which the cortex was extremely congested but showed no other lesion. In this case perivascular cuffing was observed in the pons, particularly in its dorsal part below the floor of the fourth ventricle. In this area, too, were cells which showed peripheral concentration of Nissl's granules. Unfortunately, the medulla, cerebral peduncles, and basal ganglia were not examined, but it can be said that the changes in the pons were of a surprisingly mild order: that this was not due to the short history of the case can be seen from the intense motor-cell destruction in the spinal cord.

This sparing of the cortex has been recorded several times by investigators in the United States, including the Collective Committee investigating the New York epidemic of 1907. From their evidence and other evidence at his disposal Rothman (1931) was left in doubt about the reality of polio-encephalitis. In fact, Brown *et al.* (1948) concluded from their investigation of a large series of cases of the bulbar encephalitis type that the cerebral symptoms were in a large measure due to anoxaemia, although in a few cases focal cerebral lesions were found.

Scheinker (1947) also reported no cortical involvement, but Bodian (1947), in 24 necropsies on cases of poliomyelitis, found involvement in the motor cortex alone, usually of mild or moderate degree, in 20 instances. In this series the most severe lesions were found in the brain-stem, where the substantia nigra, the cerebellar roof nuclei, the vestibular nuclei, and the reticular formation were heavily involved. Bodian felt that stupor, disorientation, and coma resulted from these brain-stem lesions, which in some of his cases included focal softening. The series also showed almost consistent involvement of the fifth, seventh, tenth, and twelfth cranial-nerve nuclei, the seventh being slightly more involved than the others. In the cord the anterior horn cells were much more involved than those of the posterior and intermediate horn cells, and were sometimes wiped out over whole segments. These findings again correspond to those in our case.

Scheinker found a distribution of lesions in the C.N.S. comparable with those found by Bodian, the medulla, pons, mid-brain, and cerebellum all being markedly involved in his cases of poliomyelitis. He also described heavy perivascular cuffing in the substantia reticularis, in the floor of the fourth ventricle, in the periaqueductal region of the brain-stem, and in the substantia nigra. But in these regions rarely was there actual destruction of the nerve cells, and complete chromatolysis was the most advanced lesion found. Lesions in the corpus striatum, the globus pallidus, and in the hypothalamic nuclei were likewise of only moderate severity.

Scheinker found numerous foci of lymphocytes in the cranial nerves, particularly in the vagi, and also an especial concentration of cells around the vagal nuclei. Apropos of these findings he quoted the view of Sabin (1942) that the virus may spread up the vagi from the alimentary tract to the central nervous system. A simultaneous cellular inflammatory reaction, including multiplication of microglial cells and degeneration of nerve cells, was a feature of Scheinker's cases. The cellular reaction was most prominent in the medulla and mid-brain, and the cell degeneration was most pronounced in the anterior horn cells of the cord.

These main lesions of fatal poliomyelitis correspond to those in the fatal encephalitic type of case described by us. In the less severe cases which recover it is probable that the lesions are of simultaneous type and distribution but of less intensity. The extensor plantar responses in our case and the spastic paresis seen during recovery in their case by Brewis and Neubauer are evidence of a pyramidal involvement, an involvement which McAlpine (1947) has described as occurring, albeit uncommonly, in the spinal form of the disease. But it is a matter for comment that, apart from occasional motor-cortical lesions in Bodian's series, upper motor neurone lesions have not been a prominent feature to the histologist: not even pyramidal demyelination was seen in our case, and no cortical or pyramidal lesions were observed by Scheinker.

It is of course possible that the intense cortical congestion in the present case was responsible for depression of upper motor neurone function, and though cord oedema was thought of little consequence by Bodian it might be associated with the transitory Babinski responses seen in some of the spinal cases. The initial flaccidity which may be associated with these responses in some cases (e.g., that of Brewis and Neubauer) was attributed by Toomey (1940) to "cord shock," a state certainly more intangible than that of congestion or oedema. As Scheinker pointed out in describing the inflammatory response of the nervous tissues, the *whole* nervous system is invaded by the virus of poliomyelitis. It is therefore not improbable that where a pyramidal lesion occurs and recovers in a more protracted case of poliomyelitis there has been some degree of demyelination of the tracts. Such changes did not develop in our case, possibly because of its short course.

A final feature of interest is in the comparison of this case with a series of cases observed in the same district (Jennings, 1947) during the spring, summer, and autumn of 1946. These cases included eight with encephalitic symptoms (apathy, twitchings, severe drowsiness, and delirium); four with facial palsies and other cranial-nerve lesions as well as cerebral symptoms; and a number of cases, some with largely meningitic signs and symptoms, in which there were temporary Babinski responses. Although only one of these cases, and that at school, had proved contact with poliomyelitis, it now seems possible, in retrospect, that at least some of them were cases of poliomyelitis in which only the brain-stem and upper motor neurones appeared to

be involved. This likelihood was mentioned by MacAlpine (1947) after the 1947 period of poliomyelitis had included similar cases; our present case, though affording no proof, does give this concept further support.

Summary

A fatal case of infection by the virus of poliomyelitis has been described.

Although the case clinically showed a mainly encephalitic and brain-stem involvement the cerebral changes were merely those of cortical congestion, and the pons showed only a mild inflammatory reaction. The spinal cord, on the other hand, showed gross and widespread destruction of anterior horn cells. No really satisfactory histological explanation was forthcoming from either cord or brain to account for the definite clinical evidence of upper motor neurone involvement. Congestion, oedema, or very slight early changes in these neurones might be adduced as causes of the signs. Inoculation of the nervous tissues of this case in monkeys resulted in symptoms and histological lesions closely akin to those in the human subject.

This seemingly encephalitic case was thus proved to be due to an infection by poliomyelitis virus, and its features are compared briefly with those shown by possible atypical cases of the same infection seen in the same district two years previously.

Our thanks are due to Miss M. H. Shaw for the photomicrographs of the sections.

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FATAL CASE OF ACUTE ENCEPHALOMYELITIS DUE TO POLIOMYELITIS VIRUS

BY

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The number of instances in which poliomyelitis virus affects the fore-brain and brain-stem seems to vary in different epidemics. Reports dealing with the 1947 epidemic in this country indicated a higher proportion than usual of cerebral and bulbar lesions with or without spinal involvement (Kelleher, 1947; McAlpine *et al.*, 1947). The picture has been confused by the occurrence, just before the poliomyelitis outbreak of 1947, of minor and localized outbreaks of virus encephalomeningitis recorded by Jennings (1947) and Laurent (1947) and regarded by them as a disease or group of diseases in which no relation to poliomyelitis was probable. On the other hand both Critchley (1948) and McAlpine *et al.* (1947) believe that the virus responsible was probably identical with that of acute poliomyelitis.

Undoubtedly the lack of facilities for the isolation and identification of virus in this country has accentuated the confusion, and, whereas Toomey (1941) recognizes eight clinical types of poliomyelitis, doubts about the existence of types other than spinal and brain-stem are held by many authorities in this country—for example, Walshe (1947), who states: "It is probable, however, that only the spinal type and a few cases of brain-stem type really belong to this [poliomyelitis] infection." Baker and his colleagues (1947), in a comprehensive review in which the word "bulbar" has been used "with some degree of licence" to denote involvement of the entire brain-stem, including the medulla, pons, and mid-brain, present an acceptable classification of all forms of bulbar poliomyelitis. They include a "diffuse encephalitic" and a "focal encephalitic" group, and, in connexion with the latter, state that aphasic disorders have been reported rather infrequently. They quote one case of visual verbal agnosia from the literature.

The following record is a description of a case of acute polio-encephalomyelitis in which during life it was not possible to make a clear clinical distinction between poliomyelitis or other virus aetiology, but in which histological evidence and ultimately virus isolation established the aetiological agent.

Case Report

A man aged 25 with a record of excellent health was admitted to the Western Hospital on Oct. 12, 1947, with a history of illness which had begun six days earlier with a sense of fatigue and malaise, followed 48 hours later by headache and giddiness. On the following day (Oct. 9) he vomited once and, because of increasing malaise and the onset of giddiness, tremors, and blurring of vision, had to take to bed. On the fourth day after the onset feverishness and thirst were pronounced. He was restless and unable to sleep, and had difficulty in hearing. His condition rapidly worsened, until on Oct. 12 he was found by his relatives to be unable to move, to be generally rigid rather than weak or flaccid, and, though conscious, to be unable to speak.

On admission at 5 p.m. on the same day he had a temperature of 101° F. (38.3° C.) and a pulse rate of 82. He was conscious, and there was some cyanosis of the extremities. He was unable to speak. A fine tremor of the outstretched hands was noted. There was moderate neck rigidity and hamstring spasm. He moved his limbs very slowly. Movements were stiff, jerky, and apparently uncoordinated, and passive movements of the limbs further revealed the general rigidity and tonic spasm. The facial muscles were involved in the general hypertonicity, and commands were obeyed incompletely and with great difficulty and delay in execution. There was fine nystagmus in all directions but most pronounced to the right. Apart from a possible weakness of the left sternomastoid muscle there were no obvious cranial-nerve palsies. All tendon reflexes were present but depressed, and the abdominal reflexes were normal. There was a doubtful Babinski sign on the right; the left plantar reflex was flexor. The respiratory muscles were unaffected and the pharynx functioned. There was no retention of urine, but he was incontinent.

A few hours later the tendon reflexes in the right lower extremity were very sluggish, but apart from the dysfunction, which was apparently due to tonic spasm, there was still no apparent paresis or paralysis; the dysarthria might also be ascribed to tonic spasm, although possibly it may have been a true motor aphasia. Lumbar puncture showed a clear fluid containing 18 lymphocytes per c.mm. and 55 mg. of protein per 100 ml.; colloidal gold test, 0111210000; Wassermann reaction negative.

On the following day (Oct. 13) there was little change, but some weakness of the right lower extremity, with greatly diminished reflexes, was noted. Consciousness appeared to be clouding and ultimately all response to stimuli failed. The tonic rigidity persisted and a fine tremor of the upper lids was

noticeable and persistent. A cinematograph record of the facies exists (Ministry of Health, 1948). A consultant neurologist made a provisional diagnosis of acute disseminated encephalomyelitis. By the next day (Oct. 14) there was marked deterioration, with a rising temperature and a tendency to accumulation of buccal secretions, but still no respiratory muscle involvement. Death, apparently due to bulbar failure, occurred rather suddenly the same evening.

Necropsy on Oct. 15 revealed a little focal haemorrhagic bronchopneumonia in the congested lungs, cloudy swelling and congestion of the liver and kidneys, and oedema and congestion of the spleen (all confirmed microscopically). The heart was normal, the air passages were clear, the pancreas and stomach were congested, and the urinary bladder was dilated. There was moderate oedema and congestion of the somewhat swollen brain and spinal cord.

Microscopical examination showed very severe perivascular "cuffing," mainly with lymphocytoid cells, and foci of acute inflammation and degeneration of parenchyma, in places almost purulent, in the basal ganglia, mid-brain, pons, and medulla—most severe in the mid-brain. The cerebral cortex in the left prefrontal, right Rolandic, and left occipital regions showed only oedema and congestion. The third and fourth cervical, fourth and eighth thoracic, and second lumbar segments of the spinal cord showed inflammation and degeneration of the grey matter, especially of the anterior horns, in which a considerable number of neurones were destroyed, and to a smaller extent of the white matter.

The findings were characteristic of acute polio-encephalomyelitis (A.B.B.).

The histological report was submitted to the consulting neurologist (Dr. T. Rowland Hill), who remarked: "A case of fulminating polio-encephalomyelitis in which, before death, it was not possible to demonstrate involvement of lower brain-stem motor nuclei. There were signs of severe mid-brain lesions in that there were tonic fits of decerebrate rigidity and marked nystagmus. There was also a degree of flaccid weakness of the right lower extremity with markedly diminished reflexes. Bulbar palsy developed before death."

Specimens of the brain and cord were stored at the Virus Reference Laboratory, Colindale, in glycerol-saline for ten months at −10° C. A 10% suspension of cord and medulla was then inoculated intracerebrally into one rhesus monkey under ether anaesthesia. The monkey developed typical signs of poliomyelitis ten days later and was killed with chloroform. Histological examination of the central nervous system showed lesions pathognomonic of poliomyelitis (F.O.MacC.).

Summary

A case is recorded of acute encephalomyelitis of clinically undetermined aetiology, in which histological findings and virus isolation established the virus of poliomyelitis as the cause.

Our thanks are due to Dr. W. H. Bradley, of the Ministry of Health, for helpful suggestions.

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Hospital matrons and nurses from Belgium, Canada, Cyprus, Denmark, Egypt, Finland, France, Greece, Italy, the Netherlands, Norway, and the U.S.A. have recently attended a two weeks' course on nursing arranged by the British Council in Edinburgh. Sir Andrew Davidson, Chief Medical Officer of the Department of Health for Scotland, gave the opening lecture.

STREPTOMYCIN IN DEVELOPMENT OF HYDROCEPHALUS IN TUBERCULOUS MENINGITIS*

BY

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[WITH PHOTOGRAVURE PLATE]

Since the introduction of streptomycin in the treatment of tuberculous meningitis several reports of hydrocephalus have appeared. Flory and his co-workers (1947) describe a case of relapse of tuberculous meningitis after cessation of streptomycin treatment in which an internal hydrocephalus was discovered at necropsy. Baggenstoss *et al.* (1947) record two fatal cases of tuberculous meningitis treated with intramuscular and subcutaneous streptomycin in which occurred heavy exudate at the base of the brain and moderate internal hydrocephalus. Wright and Rees (1948) report three cases of severe hydrocephalus associated with thick basal exudate after treatment with intramuscular and intrathecal streptomycin for at least eight weeks. Smith *et al.* (1948) describe two fatal cases with gross hydrocephalus after intramuscular, intrathecal, and intraventricular streptomycin; in two subjects who survived, ventriculography before treatment revealed some dilatation of the ventricles. In all of these cases exudate in the basal cisternae was found to be most intense.

An M.R.C. report (1948) includes 46 instances of hydrocephalus among 53 necropsies on cases of tuberculous meningitis treated with streptomycin by various routes. These studies led me, whilst interested in experimental hydrocephalus, to investigate the histological changes in the basal meninges in a small series of cases of tuberculous meningitis with the object of finding out if the administration of streptomycin had modified the lesion in any way that might lead to hydrocephalus.

Materials and Methods

The leptomeninges from the undersurface of the pons were available for histological examination from eight cases of tuberculous meningitis; six of these were hydrocephalic, two were not. Seven had been treated with intramuscular and intrathecal streptomycin, with or without intraventricular streptomycin, for periods varying from 12 to 231 days, while only one (hydrocephalic) received no antibiotic treatment. Fig. A shows the dilatation of the anterior horns of the lateral ventricles in the frontal section of the brain of a case of tuberculous meningitis treated with streptomycin.

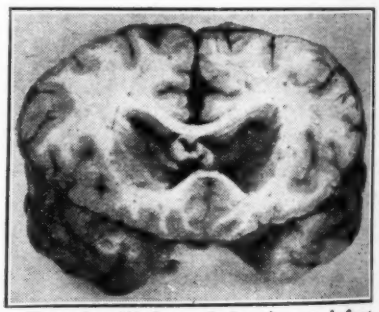

FIG. A.—Hydrocephalus in an infant following tuberculous meningitis treated with streptomycin.

The leptomeninges, with a piece of brain tissue, were cut out from the undersurface of the pons and fixed in 10%

*Part of a thesis submitted to the University of London for the Doctorate of Philosophy.

formol-saline ; the planes of section of the specimens were at right angles to the long axis of the brain. These were slowly dehydrated and embedded in paraffin, and thin sections were stained with Ehrlich's acid haematoxylin and eosin as well as with Weigert's iron haematoxylin and van Gieson's stain.

Results

The basal subarachnoid space in all the hydrocephalic cases were completely blocked by a fibrinous exudate and thick tuberculous granulation tissue (Plate, Figs. 1 and 2). Two of the five treated cases showed some fibroblastic proliferation within the exudate, mainly directed against the fibrinous elements but also partly encircling some minute tubercles (Fig. 3). The other three gave no evidence of organization, nor was there any obvious histological modification of the lesions by streptomycin when compared with the lesions from the untreated case.

In the two non-hydrocephalic cases, both of which had been treated with streptomycin, the subarachnoid channels were patent in spite of the presence of meningitis (Fig. 4). The fibrinous exudate and the tubercles were there, but they were much less prominent. The lesions did not appear to have been essentially modified, nor was there any evidence of fibroblastic proliferation.

In three of the hydrocephalic cases the foramina of Luschka were found to be patent from the outside, and in one case histological section through this region showed the lateral recesses, including the foramina, to be unobstructed by tuberculous lesions.

Discussion

Fibrosis of the tuberculous lesions is known to be a general response to streptomycin treatment both in miliary tubercles in man (Flory *et al.*, 1947 ; Baggenstoss *et al.*, 1947 ; M.R.C. Report, 1948) and in experimental lesions in guinea-pigs (Feldman *et al.*, 1945). This naturally aroused the suspicion that streptomycin, in the course of promotion of healing of tuberculous lesions in the meninges, might lead to fibrosis and adhesions, resulting in obstruction of the circulation of cerebrospinal fluid and consequent hydrocephalus.

The fact that intrathecal streptomycin had been given in many of the reported cases of hydrocephalus strengthened the view that leptomeningeal adhesions after such treatment were responsible for the blockage. But in the present investigation only two of the five streptomycin-treated cases showed some fibroblastic proliferation in the basal leptomeninges, and although this reaction did surround a few tubercles it seemed to be mainly directed along the fibrin threads in the exudate. Similarly, only one of the two hydrocephalic cases of Baggenstoss *et al.* (1947) and two of 46 hydrocephalic brains of the M.R.C. report (1948) showed any fibrosis in the meningeal exudate.

Further, Rich (1944) noted the presence of some degree of organization and repair in the meningeal lesions of the more protracted cases in pre-streptomycin days. Long ago Martin (1909) recorded scar tissue as replacing antecedent tuberculous lesions in the meninges in cases that had recovered from tuberculous meningitis, while Kramer and Stein (1931) also pointed out the tendency to fibrosis of the meningeal exudate in similar cases. These facts suggest that the formation of leptomeningeal adhesions has not always been responsible for the observed hydrocephalus, nor has it been especially brought about by treatment with streptomycin.

On the other hand, one of my cases of tuberculous meningitis which was not treated with streptomycin also showed the presence of an internal hydrocephalus. A search through the older literature suggests that tuberculous meningitis by itself has long been suspected of being associated with hydrocephalus (Barther and Rilliet, quoted by Burr and McCarthy, 1900 ; Dandy and Blackfan, 1914 ; Rich, 1944 ; Martin and Elkinton, 1947 ; Bucy, 1948). Further, it has been possible to produce internal hydrocephalus in guinea-pigs after the introduction of *Mycobacterium tuberculosis* through the cisterna magna (De, 1949). It may therefore be concluded that tuberculous meningitis alone can give rise to hydrocephalus, and the prominence given to the condition since the introduction of streptomycin treatment is no doubt due to more careful examination of the brain in such cases.

The hydrocephalus found in association with tuberculous meningitis has mostly been of the communicating type (Wright and Rees, 1948 ; Smith *et al.*, 1948 ; M.R.C. report, 1948). In some of the brains of my series the foramina of Luschka were shown from outside to be free from any obstruction. In one case histological section confirmed this naked-eye finding. These observations support the findings of previous workers that the hydrocephalus is of the communicating type. Moreover, in all the hydrocephalic cases, treated and untreated, the wide subarachnoid space at the undersurface of the pons had been fully occupied by a fibrinous and caseous exudate with tuberculous granulation tissue ; there appeared to be no room left for flow of any cerebrospinal fluid. On the other hand, the two non-hydrocephalic cases presented open subarachnoid passages in between the scanty exudate.

I have also found that the cisternal introduction of *M. tuberculosis* in guinea-pigs is followed by hydrocephalus associated with partial blockage of the cisterna pontis with considerable cellular proliferation, but in the rat this change is very slight and there is no evidence of blockage or of hydrocephalus (De, 1949). These facts also suggest that internal hydrocephalus in tuberculous meningitis is due to and occurs with obstruction of the basal subarachnoid by the tuberculous exudate, independent of any streptomycin treatment.

It should be noted in passing that in most of the present cases with streptomycin treatment the tuberculous lesions elsewhere in the body have shown some modification in the form of increase of collagen, and in one case almost complete replacement by lymphocytes. But in spite of the intrathecal administration the meningeal lesions failed to respond. Of course, in the two non-hydrocephalic cases the basal exudate was less intense, but it is difficult to decide if this is an effect of streptomycin, especially in the absence of any essential change in the histology. It is interesting to recall that the M.R.C. report (1948) recorded a similar finding in relation to the meningeal lesions where the fibrinous exudate around the lesions was suspected to shelter the bacilli from the effect of the antibiotic. Hinshaw and Feldman (1944), the originators of streptomycin therapy in tuberculosis, also realized that pathological characteristics of the local lesions can impose marked limitations in the treatment of clinical tuberculosis.

Conclusions

Tuberculous meningitis can give rise to an internal hydrocephalus associated with complete blockage of the cisterna pontis by tuberculous granulation tissue and a copious fibrinous exudate, but independent of the development of cisternal fibrosis. There is no convincing evidence that streptomycin increases the chance of this complication by stimulating the production of reparative tissue.

I wish to thank Professor G. R. Cameron, F.R.S., for his help in the preparation of this paper, and Mr. McDonald for the photomicrographs reproduced in the photogravure plate.

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Medical Memoranda

Paraffin-wax in the Male Bladder

The following case of a foreign body in the bladder, and the method of evacuating it, may be of interest.

CASE REPORT

One evening a young man, acting under an obsessive compulsive urge, fashioned a bougie, about 2½ in. (6.25 cm.) long by about ¼ in. (0.6 cm.) in diameter, out of candle droppings and passed it up his urethra, from whence it slipped into his bladder. The next morning he came to hospital complaining of pain and frequency and giving the above history. Cystoscopy showed the bougie, broken into three fragments, as three white rods floating in the water used for distending the bladder. A Braasch direct cystoscope was passed and efforts were made to grasp the fragments with a Bumpus specimen-taker. These efforts were unsuccessful, because the paraffin rods, floating at the top of the bladder, eluded capture by bobbing away. As the paraffin-wax from which this candle was made was capable of solution by liquid paraffin of medicinal quality of a specific gravity of 850 at a temperature of 120° F. (48.88° C.), it was decided to try dissolving the bougie. A piece of candle ¾ in. (1.9 cm.) long could be dissolved in 20 minutes, and it was hoped that solution of the bougie would be effected by washing out the bladder for an hour with the liquid paraffin at a temperature of 120° F. The bladder was accordingly filled with the specified liquid paraffin and emptied at two-minute intervals, 2 oz. (57 ml.) of the oil at 120° F. being used for each filling. The washings from the bladder undoubtedly contained fine particles of wax, but cystoscopy immediately afterwards showed no appreciable difference in the size of the rods. The possibility of melting them by using short-wave diathermy was inquired into, but as the elevation of temperature would not exceed 5° F. (2.8° C.) above blood heat the idea was abandoned.

In 1924 Wardill, of Newcastle, described a technique for cystoscopy using liquid paraffin as a medium instead of water. The wax from which the bougie was made sank in liquid paraffin of a specific gravity of 850, and by using liquid paraffin instead of water and thus having the fragments of the bougie lying on the trigone instead of floating near the fundus it was hoped that the Braasch cystoscope and Bumpus specimen-taker, so useful in the extraction of foreign bodies heavier than water—stones, etc.—might do the trick. However, we met with failure again, because air bubbles, which the bubble extractor of the Braasch cystoscope could not expel, prevented visualization and the bougies were not even glimpsed.

As a last resort, 4–5 oz. (114–132 ml.) of liquid paraffin of a specific gravity of 850 was instilled into the bladder and the patient was asked to void. It was hoped that this manœuvre, by bringing the fragments of the bougie more on a level with the internal urinary orifice, might effect their evacuation. At the first filling one fragment of the bougie came away, and at a subsequent filling, a couple of days later, the remaining fragments were voided. Doubtless the lubricating effect of the liquid paraffin played some small part in helping their expulsion. The above operations were carried out under penicillin-sulphonamide cover.

It is quite possible that the use of liquid paraffin of low specific gravity, as described above, may prove helpful in securing the evacuation of other lighter-than-water foreign bodies not too large to be voided. The method seems so innocuous that it is worth a trial, and information regarding such trials would be very welcome.

It is obvious that it would be simplicity itself to remove these fragments by cystotomy, but it seemed a pity to do this if it could be effected otherwise.

It is a pleasure to acknowledge help from Dr. R. Collis, Dr. Norman Moore, Mr. Albert le Brocquy, of the Greenmount Oil Co., Dublin, Dr. Dacre Hamilton, of Dublin University Medical School, and Mr. Morris, of the Irish Electro-Medical Co.

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REFERENCE

Wardhill, W. E. M. (1924). *Lancet*, **1**, 179.

Histamine Desensitization in Ménière's Disease

The following case of moderately severe Ménière's disease responded so well to treatment by histamine desensitization that it is considered worth recording. This form of treatment in histamine-sensitive patients is most strongly recommended, as there is little doubt that it achieved success in this case. The result also supports the findings and experience of Miles Atkinson and others who have obtained success in the great majority of their cases.

CASE REPORT

The patient, a high-ranking officer in the Royal Air Force, was posted abroad in November, 1946, to take command of a very important air-training project. Shortly after his arrival I was called one night to see him, as he was suffering from a sudden attack of intense vertigo, with nausea and vomiting, tinnitus, and deafness.

I arrived to find him quite prostrate, with extreme pallor, and covered with clammy sweat. He was vomiting repeatedly and complained of pronounced giddiness except in the prone position. His pulse was very rapid and barely palpable. There appeared to be a momentary loss of consciousness. I could not detect nystagmus, though this sign was evident in subsequent attacks. The cardiovascular, respiratory, and central nervous systems were clinically normal and I could find no evidence of focal sepsis. Little doubt remained that this was a case of Ménière's disease—a diagnosis supported by his previous history.

His wife informed me that he had suffered two previous attacks in England, the first some months previously and the second shortly before he left the United Kingdom. He had been seen then by two specialists, both of whom had confirmed a diagnosis of Ménière's disease. He had been taking 2 gr. (0.13 g.) of phenobarbitone twice daily for several months and had been given the usual instructions regarding reduced salt and fluid intake. I prescribed bromides and hyoscine, which gave some relief, but his attack continued practically throughout the night. He had completely recovered in 48 hours, but stated that his hearing was still somewhat impaired and that tinnitus was troublesome. It was known that he had

been suffering from a high-tone deafness for many years, and the impairment of hearing was therefore not altogether related to Ménière's disease. Exactly a fortnight later I was again called out in the early hours and found him in a precisely similar attack, from which complete recovery occurred again within two days. The attacks continued at roughly two-weekly intervals, a total of six being recorded.

In January, 1947, some two months after his arrival overseas, when I had attended him in five attacks, I was faced with the problem of deciding whether this valuable officer could remain abroad or whether I should have to invalid him. He had been specially selected for this post because of his wide experience, and he was naturally most anxious to fulfil his mission. I discussed his case with several colleagues, including an E.N.T. consultant. As a result of these discussions and information obtained from current literature—e.g., Atkinson, M., *Proc. R. Soc. Med.*, 1946, **39**, 807—a skin test was carried out in order to determine whether he was histamine-positive or histamine-negative. The test is fully described in the "Any Questions?" section of the *British Medical Journal* of May 10, 1947 (p. 666), and is of course an essential preliminary to treatment if histamine desensitization is contemplated. The patient proved to be a well-marked example of the histamine-sensitive group, and I decided that he might well benefit from a desensitizing course of histamine. This treatment is also described in the section of the *B.M.J.* quoted above.

The sixth attack since his arrival developed in the early stages of treatment, which was begun in January, 1947, but was notably less severe than those from which he had previously suffered. The course was completed in April, 1947, since when (apart from mild tinnitus), he has been in excellent health and completely free from further attacks. There has also been subjective improvement in his hearing. It should be mentioned here that he has had no phenobarbitone or other medication for the past 12 months.

As more than two years has elapsed since this treatment was first given, it is reasonable to assume that a cure has probably been effected; in any case, a remarkable response has been demonstrated to histamine desensitization. Several skin tests carried out since this time have shown a consistently negative reaction.

A point of interest is that the patient's flying category, which was reduced in 1946 to non-combatant flying (passenger only), has been restored to that of an active pilot category, and he has been able to carry on with an arduous and intricate task at a highly responsible level with the utmost confidence and efficiency.

I wish to thank the Director-General of R.A.F. Medical Services and the patient for permission to publish this case

SAVILLE PAUL, M.R.C.S., L.R.C.P.,
Principal Medical Officer, R.A.F., Southern Rhodesia.

Congenital Cyst of the Cystic Duct Containing Calculi

I have been unable to find a report of a cyst of the cystic duct; it may be of interest, therefore, to record a case that has been under my care recently.

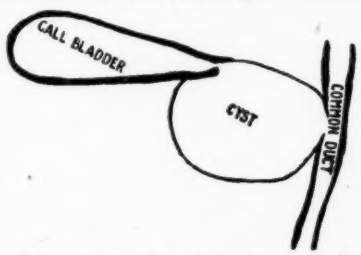
CASE HISTORY

A married woman aged 56 was referred to me on account of attacks of upper abdominal pain and vomiting for two years. During the attacks some tenderness had been elicited over the gall-bladder region. There had been no previous illness, but during this period she had had discomfort after eating, with flatulence, and had gained considerably in weight. There had never been any jaundice. The attacks of colic and vomiting had coincided, and the vomit was either green or brown. She had never been pregnant, and the menopause occurred at age 50.

The patient was very obese and thick-set, but otherwise quite healthy-looking. The tongue was clean, the pulse regular 108 a minute, and the blood pressure 110/65 mm. Hg. No physical signs were detected except some haemorrhoids. During chemical and radiographic investigations (during which the only abnormality noted was a group of five ring shadows medial to and a little below the shadow of a gall-bladder which emptied normally) the patient had two further attacks of colic, and after one of these I detected a definite tenderness below the right costal margin. In view of the severity of the attacks an exploration of the biliary passages was carried out.

Operation.—A general anaesthetic consisting of hexobarbitone, gas, oxygen, and ether by intubation was administered by Dr. E. S. M. Merry. A transverse incision was employed, but owing to the patient's obesity, the flatulence, and the high gall-bladder the incision had to be extended upwards through the rectus sheath to get the necessary exposure. The gall-bladder was partially enclosed in a tunnel of liver substance. Its fundus protruded through the anterior surface of the liver. The organ was distended with bile but did not look unhealthy; but medial to its neck a hard mass could be felt deeply buried in fat which also contained the common bile duct. On dissection of the mass the common bile duct was seen to be of normal size and free of calculi.

The hard mass was in the situation of the cystic duct and consisted of a thin-walled cyst packed tightly with calculi and biliary mud. On traction on the neck of the gall-bladder it was seen to be in continuity with the cyst (see Fig.). In an attempt to bring the parts nearer to the surface the gall-bladder was dissected from the liver, but immediately it was pulled on gently it tore away from the cyst, with which it communicated by a small aperture. This was enlarged and the contents of the cyst evacuated—the mud by suction and five black pigment calculi the size of small peas by forceps. The walls of the cyst, which was the size of a hen's egg, were extremely thin and translucent. In view of the difficulty anticipated in removing it a tube was passed into the common duct through the cyst cavity and the size of the cyst reduced by plicating sutures. The gall-bladder bed was then peritonized and the wound closed.



Examination of the gall-bladder after its removal revealed cholesterosis and three calculi similar to those in the cyst itself. As the wall of the gall-bladder was at least of normal thickness and that of the cyst was thin, translucent, and devoid of fibrous tissue and muscle, I consider that the cyst was of congenital origin and not part of an hour-glass gall-bladder. There was no inflammation, and the symptoms seemed to have arisen mechanically when its fluid contents had been replaced by the mud and calculi. These appeared to fill its cavity, but the cholecystogram showed that a passage for bile still existed between the common duct and the gall-bladder through the cyst itself. No trace of a cystic duct was seen, and the opening from the cyst into the common duct was large enough to take a No. 10 catheter easily.

COMMENT

I believe that the absence of biliary mud from the gall-bladder and common duct and its presence in the cyst was due to the flow of bile carrying into the duodenum any that escaped into the common duct during attacks of colic, and that the weight of the cyst caused valve-like actions at its junctions with the common duct and gall-bladder.

I am indebted to Professor Paterson Ross for his helpful comments in presenting this case for publication.

DOUGLAS S. PRACY, F.R.C.S.Ed.,
Honorary Surgeon, Nuneaton General Hospital.

The *Manchester Guardian* (June 30) has reported that the Bath Education Committee has had to close the city's school dental service because of lack of professional staff. The result of this will be that 8,000 school-children will be deprived of regular dental inspection.

Reviews

DEVELOPMENT OF HOSPITALS

Hospital Trends and Developments 1940–1946. Edited by Arthur C. Bachmeyer, M.D., and Gerhard Hartman, Ph.D. (Pp. 819. $5.50 or £1 10s.) New York: The Commonwealth Fund. London: Geoffrey Cumberlege. 1948.

There are more than 160 contributors to this work; a few of them, including the late Lord Dawson and Sir Frederick Menzies, are British. It is a collection from the literature made to facilitate reference by those whose work is in this field—administrators, heads of departments, and students of hospital administration. Readers of the *Journal* will note with pleasure that Lord Dawson's contribution appeared originally as a leading article (May 9, 1942, p. 573), though its form here is taken from another source. Despite the unavoidable divergences from existing conditions due to its date, there could hardly be a more informative or more stimulating contribution for readers on this side of the Atlantic. There are approving commentaries upon the article by a number of leading authorities in the U.S.A. How necessary it is to think along similar lines there may be inferred from such a sentence as this, from J. S. Falk's contribution: " . . . In most urban areas and in nearly all rural areas poverty and lack of medical care go along together." All the articles upon current trends make it obvious that in the United States opinion is veering towards a plan like our own in almost every particular, and it looks as if President Truman's recommendation to Congress for a national service may find a great deal of informed support.

Turning the pages, one finds a contribution by J. A. Hamilton with headings such as these: Haphazard Location of Hospitals; Improper Construction; Poor Distribution of Skilled Personnel; Lack of Integration of Health Agencies; Lack of Trained Management; High Cost of Hospital Care; Lack of Co-ordinated Co-operation; Conduct of Regional Surveys; Establishment of Voluntary Regional Councils; Development of Group Practice; The Health Centre; and so on. They might have been taken direct from our surveys and the commentaries upon them. Article after article repeats their substance.

Part II of the book considers administrative problems. The American machinery differs from ours, and the discussion is in parts more theoretical and academic than is customary here.

Part III concerns departments. In the section on nursing there is to be found the sentence: " . . . It would seem economically sound for hospitals to consider using both professional and practical nurses." Despite the rather unkind and no doubt unintentional antithesis, it is notable that the distinction should be made between those to whom the doctors look for technical assistance and those to whom the patient looks for personal comfort, bodily and mental—a fundamental division of function, imperfectly realized. The bibliography does not include the reports on nursing produced here. The section on the operating theatre contains a chapter on electrical hazards which it would be opportune to study in this country. There is scarcely one article in this part from which it would not be possible to cull some illuminating remark.

Part IV deals with the finance, design, and servicing of hospitals; Part V with public health, health insurance, and regional planning.

The editors have done an excellent piece of work, and their book is a worthy successor to two already well known: *The Hospital in Modern Society*, and *Hospital Care in the United States.*

E. ROCK CARLING.

RHEUMATISM IN CHILDREN

Juvenile Rheumatism. By G. E M. Scott, M.B.(Melb.), L.R.C.P.Ed., L.R.F.P.S.(Glasg.). (Pp. 163. £1 5s.) Melbourne: W. Ramsay (Surg.) Pty., Ltd. 1948.

In the present state of our knowledge of acute rheumatism information on the incidence, severity, and general clinical picture of this condition in different parts of the world is much to be desired. A monograph on the disease from Australia should thus be of considerable interest.

This handsomely produced book represents the clinical impressions of acute rheumatism in childhood formed by the author while an assistant medical officer in the Rheumatic Clinic of the Children's Hospital, Melbourne. The majority of the chapters consist almost entirely of a review of some of the current literature on the subject. Unfortunately, no new data are provided on the incidence, aetiology, or pathology, and the social aspects of the disease appear to be almost completely neglected. The chapters dealing with the clinical features are profusely illustrated with detailed reports of some of the 645 cases personally observed by the author. These confirm the already well-known fact that there is little difference in the clinical picture of acute rheumatism as seen in Australia from that observed in the northern hemisphere. Although it sets forth the author's personal views, the book unfortunately adds nothing to our knowledge of this baffling disease.

C. BRUCE PERRY.

DISEASES OF THE CHEST

A Practical Manual of Diseases of the Chest. By Maurice Davidson, M.A., M.D., F.R.C.P. Third edition. Oxford Medical Publications. (Pp. 670; 268 figures. £2 10s.) London: Geoffrey Cumberlege (Oxford University Press). 1948.

In the third edition of his well-known textbook Dr. Davidson has made a number of additions and revisions. Certain of the reproductions of radiographs have been replaced by more satisfactory ones. The basic plan of the book, however, remains unchanged. Its object is to present a readable and practical account of current practice in chest diseases. Case reports from the author's life-long experience are added in the text to illustrate practical points. There is at the end of each chapter an adequate list of references, both to more detailed textbooks on special subjects and to original papers.

Dr. Davidson's literary style is leisurely, and brings with it a reminder of more spacious days. The younger physician will find described here certain methods which he may think out-moded—for instance, the creosote chamber in bronchiectasis, and the use of atropine for pulmonary oedema. While the emphasis of the book is on practical aspects, space is not always allocated in accordance with the importance and frequency of diseases in practice; the section on asthma, for example, consists of only 22 pages out of the 660 which the volume contains. It is unfortunate that in the section on bronchiectasis the diagrams and nomenclature of bronchial anatomy published by Nelson in 1934 are reproduced, whereas in a note on the anatomy of the bronchial tree and in the section on bronchography, which has been revised with very helpful line-drawings to clarify the illustrative bronchograms, the later anatomical nomenclature of Foster-Carter and Hoyle is used.

These are minor criticisms. On the whole the book can be recommended as a reliable account of chest disease which will rarely fail to give the physician some indication of what to do when faced with practical clinical problems.

J. G. SCADDING.

MEDICINE IN THE SHETLANDS

A Shetland Parish Doctor. By Harry Pearson Taylor, J.P., M.B., C.M.(Aberd.). With foreword by T. E. Saxby, J.P., F.R.F.P.S., L.R.C.P.&S.Ed. (Pp. 136; illustrated. 10s. 6d.) Shetland: T. & J. Manson (Shetland News Office, Lerwick). 1948.

It was a lucky day for Shetland in June, 1890, when Dr. H. P. Taylor was appointed parish medical officer for the islands of Yell and Fetlar, for the job and the man fitted one another like hand and glove. One would hardly have expected it, for Taylor was born in Yorkshire, received his early education in Northumberland, and had nothing Scottish about him except his training at Aberdeen University. He had gone to the place as a locum to a sick man and had no intention of remaining,

but he spent the rest of his life there, rarely leaving the island. As he says, he fell in love with the place and never wished to leave it, in spite of temptations to do so. It certainly was not the salary which tempted him—£55 a year for Yell and £15 for Fetlar—with what he could pick up in the way of private practice among a mostly poor and hard-working population.

He had to tackle everything, generally single-handed, for his nearest medical neighbour was on another island and communications were often very difficult. But Taylor was essentially a handy man, and very bravely and ingeniously he dealt with many cases which in town life would have been sent to hospital. He had a flair for mechanics, which was sometimes exercised on the engines of ships coming to the island. He and his wife built a reflecting telescope from which they derived much pleasure.

Anyone who is inclined to patronize the "mere country doctor" would get a surprise if he read this book. There is not an atom of self-pity in it, but the long walks (at first all was done on foot or by means of a boat) in desperate weather over rough country must have been trying, and there was never any question of sending the case to another doctor. For 35 years Taylor was the doctor and friend of everybody on the two islands, and it is good to learn from a foreword by the publishers that he was appreciated and loved by everybody.

The doctors in the Highlands and Islands have always had a strenuous job—one which could appeal only to men who are country-lovers and "doctors by the grace of God." Taylor tells how much the coming of the Highland and Islands Medical Service meant to him and his colleagues in the improvement of their conditions.

There are many racy stories about the people and their customs, some of which disappeared during Dr. Taylor's time. I have no doubt the book will be treasured by many Shetlanders both resident and exiled. I sincerely hope it will have a wide circulation among people who have never seen Shetland but would like to know something about it and its people. They will be well rewarded by the tale of the life of a fine character. As for myself, the story left me with a regret that I never met Dr. Taylor. He was a credit to our profession.

The book is finely illustrated by pen-and-ink sketches and some colour plates, all the work of the author, who was evidently no mean artist.

ALFRED COX.

ABNORMAL PSYCHOLOGY

Abnormal Psychology. By James D. Page. (Pp. 441; 15 figures. $4.00 or £1.) New York and London: McGraw-Hill Book Company, Inc. 1947.

Here at last is a book properly proportioned to the needs of the doctor who sees patients outside a mental hospital, whether in general practice or in the wards and out-patient department of a general hospital. The psychoses are clearly and adequately described, but they do not dominate the book. More attention might have been paid to psychosomatic conditions and the visceral neuroses if this appeal to the profession outside the ranks of the specialist psychiatrist were to be fully met, but little complaint can be directed to either the matter or the manner of the book. It is clear, concise, and well balanced.

The author gives the impression of basing his statements throughout on a close and reasoned study of facts and figures, yet he has entirely avoided a dry statistical style. It is interesting to note how doctrines which twenty years ago seemed speculative and theoretical have stood the test of practical investigation and, at least in this book, appear as well-authenticated propositions in established psychopathology.

The opening chapters give a clear picture of the differences between normal and abnormal, of motivation and adjustment, mental mechanisms and symptoms, and the general causes of abnormal behaviour. In the rest of the book the sections on the psychoneuroses, alcohol, epilepsy, and crime are specially to be commended. The profession as a whole should do their best to read this excellent book—even surgeons would benefit by its perusal.

R. G. GORDON.

BOOKS RECEIVED

[*Review is not precluded by notice here of books recently received*]

Present Concepts of Rehabilitation in Tuberculosis. By N. C. Kiefer, M.D., M.P.H. (Pp. 398. $3.50.) New York: National Tuberculosis Association. 1948.

Schafer's Essentials of Histology. Edited by H. M. Carleton, M.A., B.Sc., D.Ph., and E. H. Leach, M.A., B.Sc. 15th ed. (Pp. 655. 25s.) London: Longmans, Green. 1949.

A Textbook of Midwifery. By W. Shaw, M.A., M.D., F.R.C.S., F.R.C.O.G. 3rd ed. (Pp. 649. 22s. 6d.) London: J. and A. Churchill. 1949.

Fundamentals of Body Mechanics and Conditioning. By M. Lee, B.S., LL.D., and M. M. Wagner, B.A., M.A. (Pp. 377. 22s. 6d.) London: W. B. Saunders. 1949.

Das EKG ABC: Eine Systematik zur Auswertung von Elektrokardiogrammen. By M. Waldhecker. (Pp. 114. M. 6.80.) Stuttgart: Georg Thieme. 1948.

Schattenseiten der Ulkuschirurgie. By P. Huber. (Pp 91. Sch. 36.) Vienna: Wilhelm Maudrich. 1949

Plastic Surgery of the Nose. By A. P. Seltzer, M.D., M.Sc., Sc.D., F.I.C.S., F.A.C.S. (Pp. 305. 72s.) London: J. B. Lippincott. 1949.

Surgery of the Hand. By S. Bunnell, M.D. 2nd ed. (Pp. 930. 100s.) London: J. B. Lippincott. 1948

Campbell's Operative Orthopedics. Edited by J. S. Speed, M.D. Vols. 1 and 2. 2nd ed. (Pp. 1,643. 150s.) London: Henry Kimpton. 1949.

Die Ermittlung des Erregungsablaufs in Ungeschadigten und Geschadigten Herzen durch Analyse des Elektrokardiogramms und des Vektordiagramms. By L. Wendt. (Pp. 84. M. 12.50.) Berlin: Akademie. 1949.

Die Diät- und Insulinbehandlung der Zuckerkrankheit. By F. Depisch. 4th ed. (Pp. 176. No price.) Vienna: Springer. 1949.

Die Gonorrhoe des Mannes. By A. Wiedmann. (Pp. 163. Sch. 36.) Vienna: Wilhelm Maudrich. 1949.

Die Asthetik der Nase. By A. Berndorfer. (Pp. 87. Sch. 21.) Vienna: Wilhelm Maudrich. 1949.

Das Zwischenhirn. By W. R. Hess. (Pp. 187. 15 francs.) Basle: Benno Schwabe. 1949.

Studies in Physical Anthropology: Early Man in the Far East. Edited by W. W. Howells. (Pp. 157. $2.00.) Detroit: American Association of Physical Anthropologists. 1949.

Studi e Ricerche sulla Manna e Mannite Naturale. By D. Gigante. (Pp. 144. No price.) Rome: Istituto Poligrafico dello Stato Libreria. 1948.

Fortschritte der Tuberkulose-Forschung und -Behandlung. By H. Birkhauser. Vols. 1 and 2. (Pp. 446 90.80 Swiss francs.) Basle: S. Karger. 1948.

Lehrbuch der Embryologie. By W. Brandt, M.D., Ph.D. (Pp. 648. 56 Swiss francs.) Basle: S. Karger. 1949.

Traitement Moderne des Brûlures. By J. Virenque and J. Sécail. (Pp. 62. No price.) Paris: Vigot. 1949.

Les Maladies Endocriniennes. By H. Curschmann (Pp. 164. No price.) Paris: Vigot. 1949

Topographische Anatomie. By O. Oertel. Revised by P. Glees. (Pp. 351. 28 Swiss francs.) Basle: S. Karger. 1949.

Your Child and You. By C. Hay-Shaw. (Pp. 157. 6s.) London: John Murray. 1949.

The Sulphonamides in General Practice. By E. D. Hoare, M.D. (Pp. 90. 5s.) London: Staples Press. 1949.

Hamlet and Oedipus. By E. Jones, M.D., F.R.C.P. (Pp. 160. 12s. 6d.) London: Victor Gollancz. 1949.

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THE GRADING OF SPECIALISTS

The results of the grading of specialists have caused great dissatisfaction in all parts of the country. Much of this dissatisfaction is due to the fact that the Assessment Committees in the various regions interpret their duties differently. Thus, in one region the criteria for grading are, to the outside observer, far stricter than those in an adjacent region. This inequality naturally causes sharp resentment and a feeling of injustice. As our correspondence columns show, some men blame the Ministry of Health for this, while others suggest that the B.M.A. or the Joint Committee should act to put matters right. It is therefore, in the first place, important to make it clear with whom the responsibility for grading lies.

In November, 1948, the Ministry of Health requested Boards of Governors and Regional Boards to set up Assessment Committees for the purpose of grading. An Assessment Committee consists of seven members: two general physicians, two general surgeons, one obstetrician, and two specialists in the branch of medicine practised by the doctor to be graded; of the two specialists one is to be nominated by the university in the region. The first five of these must have practised or be practising in the area of the Board. They are chosen for "their high professional standing and wide clinical experience and their knowledge of the Services of the area." The first point, then, is that the grading is done by the profession itself, not by the Ministry of Health, not by the Royal Colleges, and not by the B.M.A.

In its circular the Ministry of Health gave the Assessment Committees general guidance. It was stated that no general criterion of specialist status could be laid down: "a proper assessment can be arrived at only after a consideration by professional advisers of the *qualifications and experience* of each practitioner" (our italics). The circular made it clear that in many instances specialist status could be decided easily on the basis of academic qualifications backed up by experience and practice as a consultant. In the case, for example, of the general-practitioner specialist it was stressed that status could not depend solely on the possession of postgraduate qualifications: the determination of status "must take account of experience, which may of itself suffice to warrant acceptance as a specialist." It does not seem that the Ministry of Health could have given general guidance in any other terms. The guidance is based on the situation that exists. Any suggestion that grading should be done by some central mechanism would, we feel sure, be resented by the medical profession. Clearly the decision must lie in the hands of the men on the spot in the regions. That Assessment Committees in different regions will interpret their duties differently seems to be inevitable; it is equally inevitable that comparison of the results in adjacent regions will cause dissatisfaction. One way of removing dissatisfaction is to establish a system of appeal that will appear fair to the man who considers that he has not been properly graded. The appeal mechanism is now the Assessment Committee with two additional members appointed to it. The practitioner appearing before such an appeal committee may feel that it is weighted against him, and it would seem fair, therefore, if at least half of the members of the Appeal Committee were to come to the hearing of the case with unbiased minds.

There is much to be said for the suggestion that in the future it is the posts that should be graded. Only those men should be appointed to such posts who fulfil the necessary criteria; and if suitable candidates cannot be found such posts should not be filled. In this way we should be able to cut across the undesirable practice of some regions of attempting to fill specialist posts with men graded as senior hospital medical officers—an attempt to avoid responsibility for making proper payment for work done. One of the problems now is to find jobs for men of registrar status, men, that is to say, who have taken higher qualifications, have had experience as specialists, and who have been qualified for four or five years. As a result of the war it has not been possible for them to find their way gradually into specialist and consultant work, and it is a commonplace that when a hospital vacancy is advertised there is for any one post a large number of suitable applicants. This applies especially to posts for general physicians and general surgeons. It is therefore difficult to believe that, with the exception of a few specialties, there is such a dearth of suitable candidates as to excuse the financial subterfuge of trying to fill posts with senior hospital medical officers. One of the aims of the new hospital service is to see that there is available in all parts of the country adequate consultant and specialist skill. That there has been an undue concentration of such skill in large cities to which teaching schools are attached is admitted. The hospitals in the small towns have to a varying extent been staffed by men who would be unlikely to be appointed to the

staff of a teaching hospital. But these men, many of them coming under the designation of general-practitioner consultant, have secured the confidence of the local medical profession and the local community as surgeons and as consultant physicians. In time it may be assumed that when they retire their place will be taken by men and women who practise entirely as consultants or specialists, many of them, it may be hoped, recruited from outstanding general practitioners in the district. In the transitional period, which may last for years, it would be unjust to attempt to degrade such men from the positions they have hitherto held. In so far as they practise as consultants and specialists they should be remunerated as such.

It must be remembered that the Spens Committee emphasized that all specialists should be treated equally, those of outstanding ability to have their services recognized by Distinction Awards. The Ministry of Health circular made it plain that hard-and-fast criteria cannot be laid down for specialist status. What is needed is an exact definition of senior hospital medical officer. The B.M.A. is resisting all attempts to include under this category those judged on the basis of either qualification or experience to have specialist status. It is doing this by refusing to advertise in the *Journal* posts for S.H.M.O.s required to do specialist work. The Ministry of Health will not make new appointments in the grade of S.H.M.O. except within a field agreed with the Joint Committee. Nevertheless, there would seem to be a case for the grade of S.H.M.O. in mental hospitals, tuberculosis sanatoria, and fever hospitals. Could it be said, for example, that an assistant medical officer in an isolation hospital should be designated as specialist and be paid in the terms of the Spens Report? The same question may also be asked of men similarly placed in sanatoria and mental hospitals.

Government spokesmen have stressed the need for more specialists and consultants and for these to be distributed more evenly throughout the country, so that the public taking advantage of the National Health Service may have equal opportunities of obtaining specialist advice and skill. Yet young men with the necessary qualifications and experience are now finding it extremely difficult to get work to enable them adequately to support themselves and their families. Other men who have been doing such work to the satisfaction of their colleagues and the local community are being financially penalized by the decisions of Assessment Committees. This unsatisfactory situation presents a challenge both to the medical profession and to the Ministry of Health. If a solution of the problem is not soon found the public will have the right to ask why they are not being given the medical benefits promised them between 1945 and July 4, 1948.

NEW PROBLEMS OF RHEUMATISM

Study of the rheumatic diseases has been discouraging to both clinician and research worker because of the lack of pathological knowledge and the absence of proved methods of treatment, but the recent work by Kendall, Hench, Slocumb, and Polley at the Mayo Clinic, to which we have already referred,[1] has dramatically widened the field in the treatment of the rheumatic group of diseases. Those who have seen the results of the treatment of rheumatoid arthritis with "cortisone" have been left in no doubt about its efficacy. In the light of this recent American research many of the different types of rheumatic disease must be regarded from a new viewpoint, and in this issue of the *Journal* there are four papers on various aspects of the subject. Dr. Bernard Schlesinger points out the importance of studying rheumatoid arthritis in the young, in whom the condition appears, as Still's disease, in its most acute and uncomplicated form. In these cases the adenopathy, splenomegaly, and fever are more marked than in adults. If the suprarenal glands are at fault in this disease, as the new work suggests, it may be that in children the rest of the endocrine system reacts by over-production: in the case of the pituitary this might, for instance, account for the labial hirsuties and possibly other unexplained phenomena. There may also be a connexion between the pituitary and the incidence of leucopenia and, particularly, lymphopenia. It has been shown that the 11- and the 11-17-oxysteroids have some control over the lymphoid tissue, and the effect of ACTH (adrenocorticotropic hormone) can be measured by the drop in circulating eosinophils and lymphocytes.[2] As Schlesinger states, further research into the blood changes in this disease is required. In the twenty cases of Still's disease which he reports four developed jaundice, and in one of these the rheumatoid arthritis vanished. It will be recalled that the recognition by Hench of the fact that jaundice induced remissions in adult rheumatoid arthritis was one of the steps which led him to the final attack on the disease with cortisone.

Some physicians have considered that rheumatoid arthritis is a psychological disorder. The results of clinical tests with cortisone, during which any possible psychological effect was carefully eliminated by giving control substances for certain periods—with relapse in each instance—has disproved this theory. The fact that shock may precipitate the onset of this disease is significant in the light of Selye's[3] work on suprarenal exhaustion and the onset of "stress" diseases. How-

[1] *British Medical Journal*, 1949, **1**, 812, and **2**, 24.
[2] Forsham, P. H., Thorn, G. W., Prunty, F. T. G., and Hills, A. G., *J. clin. Endocrinol.*, 1948, **8**, 15.
[3] *Ann. intern. Med.*, 1948, **29**, 403.
[4] Ibid., 1946, **24**, 808.
[5] *Ann. rheum. Dis.*, 1949, **8**, 90.

ever, though rheumatoid arthritis is not a psychological disorder, there is no doubt that in treating rheumatic diseases the clinician will encounter cases which can be described, in a term coined by Hench and Boland,[4] as psychogenic rheumatism. They reported that 16–18% of the patients in U.S. Service hospitals devoted to the treatment of rheumatic diseases were suffering from musculo-skeletal psychoneuroses. A careful study, with controls, of this condition is now reported by Drs. William Tegner, Desmond O'Neill, and Anna Kaldegg. They point out that in such cases the patients described their pain in unusual terms; it was widespread, and might be represented as "pain all over." The distribution of the physical findings, which were inconstant in site, did not correspond to any recognized physical disorder. These authors confirm the American view that analgesics have no effect on the symptoms, but differed in their findings that cold and damp affected both patients and controls equally. Now that psychogenic rheumatism is recognized as an entity, diagnosis should not be difficult, for the history is bizarre and the findings are unrelated to organic disease. It is important that the correct diagnosis should be made, because such patients do not require physiotherapy, which will only perpetuate their symptoms. Some method such as group psychotherapy might be tried.

Rheumatoid arthritis is of course the most crippling but not the most common form of rheumatic disease. The majority of patients attending the rheumatism department of a hospital come with conditions which can be classified under the heading of non-articular rheumatism or fibrositis. Many clinicians deride such a diagnosis, and workers in the U.S.A. tend to disregard it. But the fact remains, as any general practitioner will confirm, that this condition forms the bulk of "rheumatism." In a paper also published in this issue Dr. W. S. C. Copeman describes research work he has undertaken on the relation of fibro-fatty tissue to "rheumatic" syndromes. Certain important facts about the anatomy and physiology of this little-studied part of the body have come to light. The discovery of the fatty herniae and the oedematous changes found in the fatty tissue in certain sites should be considered in relation to the new knowledge about the connexion between the rheumatic group of diseases and the suprarenal cortex. Of the twenty-eight steroids so far isolated from the suprarenal cortex only six are known to be active, and of these desoxycorticosterone has been studied in more detail than the others, particularly in relation to Addison's disease. It is largely concerned with electrolyte balance, and in Addison's disease there is increased excretion of sodium and chlorides and retention of potassium. This may cause increased intracellular fluid tension. Copeman points out that in the dorsal and sacral fat-pads, and in similar pads round the knee and ankle where fibrositis occurs, this may be due to oedematous distension of fat owing to an upset of the water balance. He suggests there may be an endocrine cause, and the new work strengthens this concept.

Leading rheumatologists have for years insisted that diagnosis must be more accurate if progress is to be made in the treatment of rheumatic conditions. Hench emphasized last year in a lecture given in this country the breadth and complexity of the work in this field, and he pointed out that there are about a hundred different types of arthritis and another hundred musculo-skeletal conditions called "rheumatism." In spite of the recent success of the Mayo Clinic workers with cortisone, many more careful studies, on the lines of those reported in this issue, must be made before the outstanding problems still presented by the rheumatic group of diseases can be solved.

MOSQUITO ERADICATION

The object of mosquito eradication is the total destruction of one or more species in a defined area within a limited time, the area being chosen in such a way that reinvasion is unlikely, or can be easily avoided, and work can be stopped on attainment of the object. It is therefore in the nature of capital expenditure, in contrast to the normal recurring expenditure on mosquito control. Mosquito eradication originates from techniques developed by the Rockefeller Foundation as long ago as 1928 for the destruction of *Aëdes aegypti* in the control of yellow fever. The means then used were destruction of *Aëdes* larvae by normal methods, such as the application of larvicides and the removal of breeding-places, the establishment of a thorough checking system (using the search for adult *Aëdes* as an index of effective larval destruction), and the perfection of anti-larval measures where they were shown to be inadequate by the continued presence of even single adults. This system achieved such success against the essentially urban *Aëdes* in South America that it was rapidly extended over most of the continent, and now that mosquito has been eradicated from a large part of South and Central America.

The first application of this principle to anophelines was made in 1939, when it was found that *Anopheles gambiae*, the main malaria vector of tropical Africa, had

[1] Soper, F. L., and Wilson, D. Bruce, *Anopheles gambiae in Brazil*, 1930–1940, 1943, Rockefeller Foundation, New York.
[2] *Bull. World Hlth Org.*, 1948, **1**, 309.
[3] *Trans. R. Soc. trop. Med. Hyg.*, 1949, **42**, 393.
[4] *British Medical Journal*, 1949, **1**, 767.
[5] Aziz, M., *Cyprus med. J.*, 1948, **1**, 21.
[6] *Bull. ento. Res.*, 1947, **37**, 399.
[7] *Malaria, Filariasis and Yellow Fever in British Guiana*, 1948. Medical Department, British Guiana.
[8] *Proceedings of the Fourth International Congress on Tropical Medicine and Malaria*, 1948, **1**, 850.
[9] *Bull. World Hlth Org.*, 1948, **1**, 213.

gained entry into Brazil, and after seven or eight years silent multiplication had started devastating epidemics of malaria in the country around Natal. An eradication scheme was begun by the Brazilian Government with the help of the Rockefeller Foundation.[1] It was based on the same principles—Paris green was used as a larvicide, and an elaborate checking system based on the search for adults was instituted. Within 22 months of starting this work total eradication had been achieved. Success was demonstrated by the only effective test—the cessation of all anti-larval work with the maintenance of the checking system over a period of at least one year. This scheme covered an area of roughly 5,000 sq. miles and cost $2,140,000. Though the cost was high, it was considered small in comparison with the damage *Anopheles gambiae* had been causing.

The second campaign against anophelines was carried out under almost identical conditions in Egypt during 1944–5. Overwhelming epidemics of malaria in 1942 and 1943 in the 300-km. stretch of the Nile valley south of Assiut proved to be due to *Anopheles gambiae* —a stranger to Egypt which had gained entry at some recent time. The eradication scheme was started in 1944 with the help of the Rockefeller Foundation and some of the workers from Brazil. Work began in July, and the last *Anopheles gambiae* was discovered in February, 1945. Anti-larval work was stopped six months later, and the invaded area successfully passed the test of freedom in the following year. The details of this campaign have been described by Shousha,[2] and those of a similar scheme in a small area of the Sudan by Lewis.[3] Neither of these authors gives the actual cost of the work, but it is understood that in Egypt it was high, of the same order as that in Brazil, while in the Sudan it was relatively low. These three campaigns had a marked similarity—all were against the same mosquito, which tends to choose readily discoverable breeding-places and was in each case foreign to the area and perhaps not well adapted to it, and no attempt was made to eradicate indigenous species. An even more important similarity was the fact that in Brazil and Egypt there was a feeling of impending national calamity which made the operation similar to a war campaign, to be backed with all national resources without limit.

In 1946 the Government of Cyprus decided to attempt the eradication of all species of anophelines in the island as a matter of normal policy. The campaign was referred to by Professor G. Macdonald in a letter which appeared in our correspondence columns last week. The island was more malarious than either Egypt or Brazil, and was infested by seven indigenous species of anophelines, three of which were known to carry malaria. Work was begun in the face of public apathy, or even opposition, each step having to be justified on its merits. The principles of control were the same as those in Egypt and Brazil, but a solution of D.D.T. in oil was substituted for Paris green as a larvicide and the work was carried out on a prearranged three-year schedule, the progress of which was described a few weeks ago in this *Journal* by Shelley and Aziz.[4] Some parts of the island have passed the final test of control—freedom despite cessation of anti-larval measures—but this test has yet to be applied to the areas which were treated between April, 1948, and April, 1949. It is understood that one or two positive records have recently been made, and more may be expected during the remainder of this year; but this should not undermine faith in the success of this more ambitious scheme, which has been carried out at a much lower cost—a sum not exceeding £310,000, or 14s. per head of the population. As with other territories, the risk of re-invasion has to be guarded against, and the fact that it has actually happened in the vicinity of the airport[5] during 1948 has made it clear that at the next revision of the International Conventions for Aerial Navigation some further provision for the disinsectization of aircraft must be made.

These schemes might be described as of the "old type," in that they do not necessarily make use of the newer methods of control. The new type, utilizing them as a matter of principle, was initiated in British Guiana, where Symes and Hadaway[6] carried out the experimental work; this was later enlarged by the Government into an executive scheme under the direction of Giglioli, who has since reported on its outcome.[7] Though the country is large, 90.5% of the 375,000 inhabitants are concentrated in a narrow waterlogged coastal strip infested by numerous mosquitoes, of which *A. darlingi* is the main malaria vector and *A. aquasalis* a minor one. No anti-larval measures have been used, entire reliance being placed on the use of D.D.T. residual indoor sprays at intervals of eight months. In the initial stages, when only a small proportion of houses were treated, no effect on breeding was seen, but when all houses came under treatment it was observed that control was so effective that the breeding of *A. darlingi* stopped, and this species is no longer present in the populated area. As this mosquito continues to exist in the neighbouring forest the risk of re-invasion is clearly great, and can be guarded against only by the continued treatment of the populated area. The annual cost of this work per head is $0.637. It has been brilliantly successful, but it must be remembered that conditions proved unusually favourable for a residual insecticide in that *A. darlingi* shelters exclusively in houses which are of a type peculiarly easy to treat.

In 1946 the Italian Health Commission, at the instigation of the U.N.R.R.A. Italian Mission, decided to attempt the eradication of all anophelines from Sardinia by the use of residual insecticides. This scheme has not yet been reported on officially. Conditions in Sardinia resemble those in Cyprus, except that the former is

larger. It is known that the attempt at eradication solely by residual insecticides has failed, and that the old methods of larval control as used in Cyprus have now been undertaken in addition to the new ones. It seems that success will probably be achieved, but at about 10 times the cost per head of the Cyprus work. (Press reports state that the governors of the two islands had an even-odds bet of 100 litres of wine on the results of their race. The odds on Cyprus should now lengthen.) A similar scheme has just been started in Mauritius, a highly malarious island which has had severe epidemics of malaria for the past 70 years and in which the disease is carried by *A. funestus*, with *A. gambiae* as a subsidiary vector. The intention of this scheme is to attempt eradication by the use of residual insecticides and to gain data applicable to other territories in the process; larvicides will be used as a supplement if there should be no early promise of success by the intended means. This work is now in its very early stages and has not yet been reported.

Eradication techniques excited much interest at the Fourth International Congresses on Tropical Medicine and Malaria in 1948, at which Soper[8] assessed their potentialities. It was clear that they are limited, there being no prospect of success against, for instance, a forest-haunting mosquito in Central Africa; but they have not yet been fully investigated, and the WHO Expert Committee on Malaria[9] has decided as a matter of research to foster an attempt at eradication of an indigenous anopheline from a mainland territory with no natural boundaries, creating artificial boundaries in their place. The relative values of eradication as a capital work and control by the newer methods as recurrent work have yet to be decided, but it can be said that the Cyprus scheme is a pioneer one in the development of eradication of indigenous species.

CONSULTANTS ADVISED TO SIGN

The Joint Committee on Monday of this week once more discussed with the officials of the Ministry of Health the question of arbitration. As a result of that meeting the Committee has decided to advise consultants and specialists to enter into permanent contracts. It was assured that no changes will be made in the terms and conditions of service without prior discussion in the medical section which is to be established as part of the Whitley machinery. The Ministry of Health agrees that remuneration is a subject on which there may be arbitration in the event of disagreement. The Whitley Councils, it should be clear, provide a mechanism for conciliation. If, for example, it is not possible to reach agreement on remuneration the matter can—save in exceptional circumstances—go to arbitration by common consent of the Minister of Health and of the medical profession. "Exceptional circumstances" means such events as a grave economic crisis. If only one of the parties is prepared to go to arbitration the matter can still be referred to a committee, to inquire into and report on the dispute, no doubt with recommendations.

It was made plain in the Committee Stage of the Amendment Bill that no party in the House of Commons will allow either side to compel arbitration. The Joint Committee may be congratulated on having secured from the Ministry agreement that remuneration is open to arbitration, and it is reasonable to assume that in the event of serious disagreement neither the Minister of Health nor the medical profession would in practice fail to seek the obvious and fair method of resolving a dispute—arbitration or a committee of inquiry. The Ministry of Health has agreed to delete from the opening paragraph of the contract the words "the Minister of Health." The Ministry, too, has agreed that patients admitted to private wards under Section 5 of the Act will pay professional fees in addition to maintenance charges, except where a public patient is admitted to a private bed for medical reasons.

In the light of the discussion in the Committee Stage of the Amendment Bill, which has been fully reported and commented on in this *Journal*, it is clear that substantial progress has been made. The question of arbitration affects other sections of the profession, notably general practitioners and those in the Public Health Service; no doubt they will now review their position.

POLIO-ENCEPHALITIS

There is at present an increase in the number of notifications of poliomyelitis. This is partly seasonal, but the figures indicate a greater rise than usual. With the increase in poliomyelitis, cases of encephalitis which must be attributed to the same virus are bound to occur. In the 1947 epidemic there was a higher proportion than usual of cerebral bulbar lesions with or without spinal involvement. When this happens during a poliomyelitis epidemic the diagnosis is easy, but at other times the aetiology can only be suspected. In 1947 Jennings[1] reported a small series of cases with encephalomeningitic symptoms which he believed were distinct from those of polio-encephalitis, and at the same time Laurent[2] described additional cases which he placed in the same unknown group. This view was criticized by McAlpine and his colleagues,[3] who concluded that in this country a brain-stem encephalitis, whether sporadic or occurring in small groups, is almost certainly due to the virus of poliomyelitis. Critchley[4] agreed with this opinion.

In this issue of the *Journal* case reports by Drs. G. H. Jennings, J. L. Hamilton-Paterson, and F. O. MacCallum, and by Drs. W. H. Kelleher, A. B. Bratton, and F. O. MacCallum, provide convincing proof of the identity of this type of encephalitis with poliomyelitis. This would probably have been decided some time ago if the necessary laboratory animals had been available: now that the wartime and immediate post-war scarcity is over and monkeys

[1] *Lancet*, 1947, 1, 471.
[2] *Proc. R. Soc. Med.*, 1947, 40, 927.
[3] *British Medical Journal*, 1947, 2, 1019
[4] *Medical Annual*, 1948, Bristol, p. 257.

can be used for research purposes, the identity of the causal agent can be established. Future puzzling cases of encephalitis can be investigated in the same fashion. The pathological pictures in the two cases described are very much alike and correspond closely to the American descriptions. Though the spinal signs may be very slight, the most severe lesions occur in the cervical and lumbar enlargements of the cord. The brain stem is affected to a lesser extent, and the cortex and subcortex show only congestion or oedema. In spite of the paucity of pathological findings the clinical signs are obvious—apathy, twitchings, severe drowsiness, delirium, and cranial nerve palsies. Death may be due to acute bulbar failure. Jennings and his colleagues comment that the pathologist finds little to support the clinical picture of pyramidal involvement. It is probable, however, that what are relatively crude techniques may reveal little change if the signs are transient or if the patient dies at an early stage of the disease. Gross, though temporary, change in function can occur in other conditions, such as the acute post-concussion state or the post-epileptic state, with nothing or very little indeed to be found by the pathologist. An interesting clinical feature of the encephalitic cases is nystagmus—a relatively common finding—and that wild type of eye movement associated with difficulty of fixation known as opsoclonia. This suggests strongly that the median longitudinal bundle is affected in the brain stem. When recovery takes place from the encephalitic type of disease it is usually complete. This is to be expected from the mild pathological changes. The fact that death is due to bulbar failure, often of the respiratory centre, suggests that if these patients can be kept alive in a mechanical respirator till the acute phase is over their chances of survival are good. If, however, there is a failure of the vasomotor centre and the cardiac centre, then it is very unlikely that any therapeutic measure will succeed.

THE CHEMISTRY OF INSULIN

Interest in the chemical structure of insulin has been stimulated by the recent work of Dodds and his colleagues on the action of enzymes on the hormone[1 2 3] and that of Sanger[4 5] on its oxidation. The successful preparation of active and serviceable pancreatic extracts by Banting and Best in 1921 and the crystallization of the hormone by Abel and his colleagues in 1926 paved the way for the chemical investigation of the molecule. The protein nature of insulin was soon established, and studies were begun on the size of the molecule and the nature of the groups responsible for the specific hypoglycaemic effect. Behind much of this work was the hope that it might lead to a clearer knowledge of the mode of action of the hormone and—perhaps even more important—might make possible a laboratory synthesis of some relatively simple "modified" or "substitute" insulin. From all these chemical studies much of real value has been learned, but a satisfactory simple substitute or a synthetic insulin has not yet been found.

There is no evidence as yet of the presence in insulin of any amino-acid or grouping not normally present in other proteins. In dilute solution at *p*H 7–7.5 it has a molecular weight of about 48,000.[6] Treatment of the hormone with a variety of chemical reagents has shown that the disulphide linkages and the phenolic hydroxyl and the primary amino-groups in insulin are necessary for its pharmacological action. From these and other chemical studies it can be concluded that the hypoglycaemic action of insulin is due not to certain prosthetic groups or to some as yet unknown amino-acid in the hormone but to a special spatial arrangement of the ordinary amino-acids. This does not exclude the possibility that the characteristic effect on carbohydrate metabolism might be due to the activity of some fraction of the molecule—i.e., by a "submolecule"—of insulin. Sanger[7] has suggested that the submolecule of insulin has a molecular weight of about 12,000 and that it consists of four peptide chains bound together by –S–S– linkages; two of these chains have terminal glycine and two have terminal phenylalanine groups. Oxidation of the disulphide cross-linkages of insulin by performic acid gives the separate polypeptide chains, and a start has been made with the work of locating the various amino-acids in these polypeptides.[4 5]

Professor Dodds and his colleagues selected insulin as specially suitable for a general study of protein structure and the action of proteolytic enzymes on the protein molecule. When pepsin acts on insulin at *p*H 2 fragments are produced which have an average chain length of about seven amino-acid residues and a mean molecular weight of about 3,000.[1 3] Purified trypsin, unlike ordinary preparations of trypsin, has only a slight hydrolytic and inactivating effect.[2] Chymotrypsin, however, removes small peptides from the insulin molecule, leaving a more massive "core," which has a molecular weight of about 4,000 and apparently consists of the slightly shortened insulin chains united by –S–S– linkages. Further information about the nature of this insulin core will be awaited with interest.

The chemistry and mode of action of insulin are being investigated by many other methods. Vesicant-treated insulin, prepared by the use of compounds resembling mustard gas and containing radioactive sulphur, has been studied by du Vigneaud and his colleagues,[8] and Woolley[9 10] has described the separation of various peptides from enzymically hydrolysed insulin. The important observations of Dr. and Mrs. Cori and their colleagues have opened another field for research. They have shown that the *in vitro* inhibition of hexokinase (an enzyme which catalyses the formation of glucose-6-phosphate from glucose in muscle, etc.) by certain freshly prepared anterior pituitary extracts is neutralized by the addition of insulin to the system.[11] It is certainly too early to give a structural formula to the "key" part of the insulin molecule, but research during the past few years has undoubtedly brought nearer this desirable achievement.

Sir Cecil Wakeley, K.B.E., C.B., F.R.C.S., F.R.A.C.S., F.A.C.S., was elected President of the Royal College of Surgeons of England on July 14, in succession to Lord Webb-Johnson, President since 1941.

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[1] Butler, J. A. V., Phillips, D. M. P., and Stephen, J. M. L., *Nature, Lond.*, 1948, **162**, 418.
[2] *Biochem. J.*, 1948, **42**, 116, and 1949, **44**, 224.
[3] Ibid., 1948, **42**, 122.
[4] *Nature, Lond.*, 1947, **160**, 295, and 1948, **162**, 491.
[5] *Biochem J.*, 1949, **44**, 126.
[6] Gutfreund, H., *Biochem. J.*, 1948, **42**, 156 and 544.
[7] Ibid., 1945, **39**, 507.
[8] *J. Amer. chem. Soc.*, 1948, **70**, 2547, 2551, and 2554.
[9] *J. biol. Chem.*, 1947, **171**, 443.
[10] *Fed. Proc.*, 1948, **7**, 200.
[11] See reviews by Young, F. G., *Science Progress*, 1948, **36**, 13, and *Lancet*, 1948, **2**, 955.

FIG. 1.—Entering the decompressor.

HIGH FLYING AND DECOMPRESSION TREATMENT OF WHOOPING-COUGH

BY

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In 1927 an air pilot of Strasbourg took his child suffering from whooping-cough for a flight to a height of 10,000 feet (3,050 metres). The child's cough was said to have ceased dramatically after three days. Before the war Dr. W. Matter (1946), of Strasbourg, did much pioneer work on aeroplane flights for whooping-cough, and the practice became popular in many countries of Europe and to some extent in South America. As a result of this and of some work done in Switzerland (P. Lauener and E. Maeder, 1942) during the war, several conclusions were reached : (1) that the best results were obtained in the fifth and sixth weeks of the disease ; (2) that several flights were no better than one ; (3) that an altitude greater than 10,000 feet was rarely necessary ; (4) that the extreme cold at this altitude was harmful to some patients, and that for this reason the closed plane was better than the open one.

Claims for the Treatment

In 1939 a controlled experiment in Berlin on 88 children treated by aeroplane flights and 33 children treated under similar atmospheric pressures in the decompression chamber yielded slightly superior results for the latter, and in addition none were made worse by the decompression treatment. The highest claims made for the treatment about the year 1945 were : approximately 30% cured—that is, cessation of paroxysms within four days ; 30% improved—that is, marked diminution of the paroxysms within seven to 10 days ; and 40% no change or worse after seven days. At the same time it was generally agreed that the beneficial effect of the treatment depended upon low barometric pressure and low oxygen tension in the alveolar air rather than upon the actual purity of the air breathed. These results were statistically open to considerable criticism owing to the absence of controlled studies and the difficulties of assessment of cure and improvement in out-patients.

In 1946 Baldry and Richou published their first report on 300 cases treated in a decompression chamber in Paris. Selected cases of all ages from 2 months to 40 years were treated, and of those followed up for 21 days (44% of the total) there were 22% of cures in four days, 51% "improved," and 4% whose paroxysms were aggravated for some days after the treatment. Up to 1948 Richou had treated some 6,000 cases in Paris. His published conclusions include the following principles : (1) that the results obtained were independent of age and weight ; (2) that the best time for the treatment is after the third week of the disease, preferably the fourth, fifth, or sixth ; (3) that repetition of the treatment does not increase the percentage of successes—that is, a good result is obtained with the first treatment or not at all ; (4) that contra-indications for the treatment are fever, respiratory and cardiac complications, epistaxis and any haemorrhage, nasopharyngeal infections, ear infections, hernia, surgical emphysema, and malnutrition. The rarefied air-pressure is regarded as the chief factor in a beneficial result. How it acts is unknown. It has been suggested that it may lead to a relaxation of bronchial muscle, damping down of the cough reflex, or alteration of the bronchial mucosa in some unknown way.

The method has its severe critics. In 1948 Bergquist treated 45 children in Stockholm—20 by aeroplane at 11,000 feet (3,350 metres) for 45 minutes, and 25 at similar low pressure in a chamber for 45 minutes. He stated that an analysis of the course of the disease, the frequency of paroxysms and vomiting, duration of the disease, bacillary findings, and changes in the blood picture showed no significant difference, and in his opinion flying and low-pressure chambers were completely without value in treatment of whooping-cough.

It is clear that a critical assessment of the treatment is required, preferably in in-patients, with radiological and haematological observations before and after. This is not easy to arrange, and to a large extent it will probably be

FIG. 2.—Inside the decompression chamber. Note the observation windows for use of doctor and technician, the telephone, the thermometer, and oxygen masks (for use in emergency only).

necessary to conduct observations on out-patients. One point that has not been established is the effect of the treatment on the various degrees of pulmonary collapse which so often complicate cases of whooping-cough in the later stages of the disease and which may not be obvious on ordinary physical examination. It is certain, however, that if there are any accompanying acute symptoms such as fever or cyanosis, or any signs of acute bronchitis, the treatment is contraindicated.

Conclusions

The conclusion at present from the work done is that the treatment is not a cure for whooping-cough in its early stages or for complicated whooping-cough. Its object is to clear up the persistent cough and vomiting of the later stages (e.g., the fourth to seventh weeks) in otherwise uncomplicated cases. Its success has still to be confirmed, and, in any case, seems to be limited. Trials are now proceeding at Park Hospital, where a renovated ex-R.A.F. decompression chamber has recently come into use. Of the 32 cases so far treated at the hospital, reports have been received on 22. In four cases the coughing had virtually ceased within four days and no vomiting occurred after the treatment; three cases were markedly improved, with very little cough and no vomiting seven days after treatment; and in 15 cases there was gradual improvement or no change. In one of the "cured" cases a symptomless right-middle-lobe collapse had completely re-expanded within seven days.

Public transport should not be used for cases of whooping-cough during the infectious stage, which is generally held to include only the first five weeks from onset of the disease, even although cough, whoop, and vomiting continue beyond that time. Applications for treatment should normally be made by doctors to the Emergency Bed Service, 10, Old Jewry, E.C.2 (Tel., Monarch 3000).

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COMMONWEALTH AND EMPIRE TUBERCULOSIS CONFERENCE

PROBLEMS OF RESETTLEMENT

The main sessions of the second Commonwealth and Empire Health and Tuberculosis Conference were reported in the *Journal* of July 16 (p. 150). One of the closing sessions was devoted to psychological and social readaptation.

Dr. W. E. CHIESMAN (Medical Adviser to the Treasury) spoke of the difficulties which attended re-employment. In a group like the Civil Service such cases were numbered in hundreds. In 1948, with a working population of 300,000, there were between 500 and 600 primary and relapsed cases of tuberculosis. He had been surprised to find the difference of opinion which existed about the length of time for which a patient required treatment. Sometimes a man was discharged from hospital and told he could return to work almost immediately, while the man's own doctor recommended another six months' convalescence. The criteria of fitness to return to work needed clarification. Another point was that the returned patient might have a certain effect on his fellow workers. General propaganda had developed a fear of tuberculosis and an awareness of the disease, and it was natural that both employers and fellow-employees should desire reassurance. A satisfactory answer to these questions would clear up some of the main difficulties of rehabilitation. It was surely not unreasonable to employ persons whom the chest physician considered to be safe, but the follow-up ought to be improved by asking the patient to get an assurance from his doctor at regular intervals that he was not a potential danger to his associates. If there was a risk from tuberculous patients let it be assessed as accurately as possible, because the future policy for the re-employment of these people must depend upon striking a proper balance between what was good for the patient and what was safe for the community.

A number of industrialists and trade union leaders took part in the discussion on this subject. A description of work in a rheumatic unit was given by Dr. J. F. BACH (St. Stephen's Hospital, London), who said, incidentally, that he believed that happy and contented people did not develop rheumatoid arthritis. Chronic anxiety and shock immediately preceded the onset so often that he felt these must be trigger factors. In his clinic patients were selected for in-patient treatment according to the assessment of personality rather than according to the degree of joint deformity.

Dr. LEWIS MOORMAN (Oklahoma State Medical Association) discussed the psychology of the doctor–patient relationship in the readaptation to industry of sufferers from chronic disease. All good doctors who managed cases of tuberculosis became good psychologists and psychiatrists. A physician should always impress on his patient the importance of the partnership between patient and doctor in the difficult task of getting well.

Dr. TREVOR LLOYD DAVIES (Boots Pure Drug Company) said that the great difficulty about special centres for rehabilitation was that it was impossible to re-create the social relationship as it existed in normal industry. He realized that it was necessary to have these centres in order to get the patient back to industry, but at the very earliest opportunity the patient should return to normal industry. Something to the same effect was said by Miss OLWEN TAYLOR DAVIES, welfare liaison officer at Papworth Village Settlement. All the schemes developed under the Disabled Persons Employment Act, as well as voluntary schemes, she said, were satisfactory only in so far as they restored the individual to economic independence and a normal life. The segregation of unfit workers was not likely to lead to complete readaptation. Experience at Papworth had shown that the patients most easily lost their sense of inferiority when working with fit people.

Infected Milk

At the final session of the Conference Dr. EDITH SUMMERSKILL, M.P., Parliamentary Secretary to the Ministry of Food, said no Government could ignore the danger to the health of the community arising from infected milk. The ultimate objective was to secure that all milk sold for domestic consumption should be in closed containers, fitted with overlapping caps to prevent the contamination of the mouth of the bottle, and that the milk should be tuberculin-tested, pasteurized, or sterilized. Unfortunately, this new policy could not be applied immediately to the country as a whole because of insufficiency of pasteurizing and bottling plants. The makers of these plants were so inundated with orders that it would take a long time to complete them. It was therefore proposed to apply the restrictions first to groups of large urban areas, and then to extend them so far as practicable to rural areas. It was probable that some twelve months would elapse before any area would be specified for this purpose, but it was hoped to complete the specifying of all urban areas within five years and most, if not all, of the rural areas within ten years.

Mr. J. N. RITCHIE (Ministry of Agriculture and Fisheries) said that at the end of May last there were 39,997 attested herds, but progress was not equal throughout the country, and the greatest progress was not necessarily attained where the incidence was originally low. The number of cattle in tubercle-free herds (attested and T.T.) in Great Britain was approximately 1,780,000. The sale of tubercle-free milk to Milk Marketing Boards for the year ended March 31, 1948, was 354 million gallons, compared with 141.6 million gallons for the year ended March 31, 1943.

According to a report in the *News Chronicle* (July 1), the health committee of the Marylebone Metropolitan Borough Council has decided to press for revision of the exemption of Government departments and Crown properties from inspection by officers of a local authority. The medical officer of health is said to have been denied access to inspect staff canteens in Government departments.

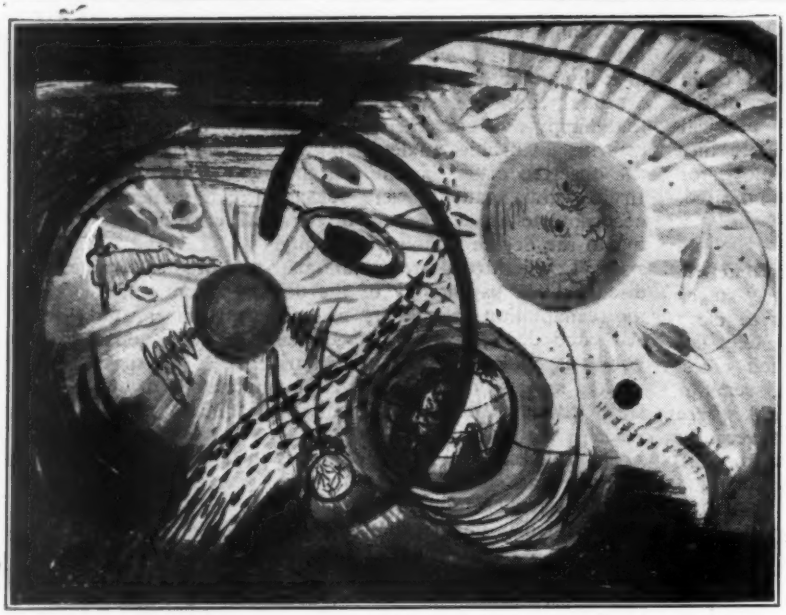

FIG. 1.—A schizophrenic patient's drawing of two universes—the real intermingling with the unreal. A typical expression of thought disturbance.

PSYCHIATRY AND ART

NETHERNE HOSPITAL EXHIBITION

For the last three years a number of patients at Netherne Psychiatric Hospital have been encouraged to paint pictures in the course of their treatment, and an exhibition of some of their productions was held at 1, Grosvenor Crescent, London, S.W.1, on July 11–16. Though nearly all the work was by people untrained in any of the arts, much of it was remarkable for the high quality of its aesthetic expression. Many of the paintings revealed vigour, imagination, and depth of feeling, and some of them were reminiscent of the products of surrealism.

A professional artist, Mr. E. J. Adamson, is attached to the hospital to help the patients overcome the difficulties of expressing themselves in an unfamiliar technique, but he leaves the choice of subjects and the actual painting entirely to the patients themselves. They paint how and what they like, but if one of them wants to know, for example, how to draw a man sitting down Mr. Adamson shows them on a separate piece of paper.

Discussing the pictures on July 14, Dr. E. Cunningham Dax, medical superintendent of the hospital, said he did not think they should use the term "art therapy." There were nearly as many "therapies" in psychiatric nomenclature as there were "phobias," and it brought only disrepute on their experimental methods of attacking psychiatric illness if they were labelled as curative without statistical evidence to support their claims. A better adjustment appeared to be made, however, when facilities were given for artistic expression rather than for some form of occupational diversion.

Painting might provide emotional release. He had seen many patients reflect their state of mind in their drawings, and apparently they felt all the better for it. The translation of emotions into paintings might give valuable information to the psychiatrist, and evidence of a patient's progress might be obtained from his paintings without ever having to discuss them. Painting might cut down the time required for psychotherapy, since some patients expressed themselves more easily on paper than in speech, and came more quickly to the interpretation in that way. Useful data might be accumulated in time on which to base the study of mental functions.

In reply to a question, he said that patients often did their most interesting work when their minds were disturbed.

FIG. 2.—Entitled "Housework," this is one of several hundred pictures drawn by a psychopathic girl who was stabilized through her work in the studio group. Since discharge she has worked for two years as a Land Girl, though previously she had never held a job for more than three weeks.

FIG. 3.—Entitled "The Lizard," this was painted by a woman with an anxiety state and some paranoid features. The patient was feeling like the lizard, which appears in the centre.

THE ROCKEFELLER FOUNDATION

POPULATION PROBLEMS

The Rockefeller Foundation last year appropriated nearly 15 million dollars for public health projects, medical research, and studies in the natural and social sciences. The new president of the Foundation, Mr. Chester I. Barnard, who succeeded Mr. Raymond Fosdick, suggests in his latest annual review[1] that in future wider fields of study may be contemplated, including some of those hitherto regarded as the preserve of religion and philosophy. There is need, he says, for a reclassification of the problems of modern society, and especially those population problems which often underlie national, class, and racial conflicts. They lend themselves to generalization and oversimplification, with quack remedies as a natural consequence. The problems are complicated by wide differences in customs, rules of inheritance, moral ideas, and religious principles. It is customary to say that the world is over-populated: "About half the world's population," says this review, "is close to starvation, and another quarter suffers from malnutrition." But the world may not be over-populated if its peoples will co-operate in the economic use of natural resources and if productive capacity can be increased. The obstacles to what Mr. Barnard calls social engineering are human inertia and insufficiency of knowledge.

Research work in genetics, endocrinology, the physiology of reproduction, and sexual behaviour has already been supported by the Foundation. The studies of Kinsey and others have emphasized the biological maladjustment between the sexual drive and population needs. The bearing of this on morals, on the incidence of venereal diseases, on frustration, and on crime is evident. In the field of human behaviour the rapid changes introduced by modern technology raise many questions. Do these changes challenge the meaning and relevancy of ancient moral precepts? What is the bearing of endocrinology or pathology on the moral assessment of human conduct? What is the relevancy of the application of ancient codes of individual behaviour to modern corporate action? The Rockefeller Foundation is proposing an examination of the nature of the conflicts of ethical systems to-day in the light of modern changes. No doubt it will find that these subjects of inquiry are much more controversial than investigations concerned only with public health and medical science. But the Foundation is taking this course because it believes that there can be no satisfactory solution to the problems of our civilization which does not take into account the ideals and spiritual aspirations of men.

WESTMINSTER MEDICAL SCHOOL

CENTENARY CELEBRATIONS

Westminster Medical School celebrated its centenary with a meeting of the staff and students held in Church House on July 13. The Earl of Athlone, Chancellor of the University of London, gave the opening address. The Dean of Westminster, Dr. Logan, Principal of London University, Lord Nathan, chairman of Westminster Hospital, and Dr. E. R. Boland, Dean of the Faculty of Medicine, London University, were among the guests. Also present were two former deans of the Medical School—Sir Ernest Rock Carling, who held office from 1909 to 1912, and Sir Adolphe Abrahams, who was dean from 1934 to 1943. Mr. H. M. Clowes, chairman of the Medical School Committee and Council, was in the chair.

Lord Athlone said that the formal connexion between Westminster Hospital and the Medical School dated only from 1849, but a medical school had existed as an offshoot of the hospital as early as 1834. He was glad to say that ties between the school and the hospital remained as close as ever, although the Medical School was part of the University of London and enjoyed a separate legal existence, with its own council and academic board. He disclosed that plans were already being considered for erecting new buildings and founding a preclinical school, although the present buildings were opened in Horseferry Road only 11 years ago. The support of the University was assured for this scheme of development.

There were at present 230 students, and the main difficulty was not to attract more undergraduates, as had been necessary in the days after the 1914–18 war, but to devise means of discriminating among hundreds of eligible and desirable applicants.

Sir Ernest Rock Carling spoke of the early history of the Medical School. He said that preclinical subjects were formerly taught, but were given up because the number of students was so small. He recalled that the Westminster Hospital was the first to build aseptic operating theatres in London, the first to make use of radium in treatment, and one of the first to employ an almoner. Sir Adolphe Abrahams discussed the advantages of the small school, where a greater number of men from other hospitals could be included in the staff.

Mr. G. H. Macnab, the present Dean, spoke of the future of the Westminster Hospital and Medical School, the difficulties in selecting students, and the pains taken by the authorities to choose the right men and women.

THE OSLER CLUB

The annual dinner of the Osler Club, held at the Royal College of Surgeons of England on July 12, celebrated this year both the centenary of the birth of Sir William Osler and its own coming-of-age. To mark the occasion several distinguished guests were invited, including their Excellencies the Chinese and Italian Ambassadors and the Austrian Minister, Dr. R. Gottlieb (representing the High Commissioner for Canada), Dr. A. S. Osborne, of the U.S. Public Health Service (representing the American Ambassador), Mr. Gilbert Frankau, Dr. Wu Lien Teh, the distinguished Chinese medical historian and organizer of the Plague Prevention Service in Manchuria, Dr. Lewis J. Moorman, editor of the *Journal of the Oklahoma State Medical Association*, and Mr. Geoffrey Cumberlege, of the Oxford University Press, which has made possible the publication of a volume of selected writings of Osler, to appear in 1950.

The chair was taken by Dr. A. White Franklin (an original member of the Osler Club), who outlined its future objects. During the dinner, speakers were introduced by Dr. W. R. Bett (another original member). The Chinese Ambassador spoke of Osler as an international physician, and the Italian Ambassador recalled Osler's visits to Italy both as medical man and as bibliophile. The Austrian Minister reminded his listeners that Osler had rounded off his medical education with a visit to Vienna.

After dinner the Oslerian Oration was delivered by Mr. Gilbert Frankau, who, although professing his ignorance of medicine, made an amusing and provocative comparison between the state of medical knowledge and public health in 1849 and the present day. He believed that the renaissance of medicine which had coincided with Osler's early years as a physician had contributed materially to his greatness, for he had taken advantage of the great opportunities open to him.

Dr. Moorman spoke of the reverence with which Osler was still regarded by American physicians, who, he claimed, knew him better than anyone else, for he had been with them longer than with the British. In Oklahoma, Dr. Moorman's home city, an Osler Society flourished, and an Osler building provided offices for a number of medical men. Dr. Moorman concluded by presenting several documents to add to the collection of Osleriana possessed by the Osler Club.

EPSOM COLLEGE

The Royal Medical Foundation of Epsom College announces that the following pensions, scholarships, and grants are now available:

A "Hadlow" Pension of £70 per annum for a medical practitioner or the widow of a medical man fully 60 years of age.

One or two St. Anne's Scholarships for girls attending Church of England schools. Candidates must be fully 9 years of

[1] *The Rockefeller Foundation.* 1949. New York.

age and the orphan daughters of medical men who have been in independent practice in England or Wales for not less than five years. The value of each scholarship is dependent upon the means of the applicant and the locality and fees of the school selected.

Pensions for necessitous medical men fully 55 years of age, provided they have been registered for five years; for the widows of medical practitioners fully 60 years of age; and for the spinster daughters of medical practitioners of 55 years and upwards. (Awards are made as vacancies occur.)

Scholarships and grants for children of either sex, not necessarily orphans. Candidates must be of public-school age and in need of such help.

Grants from the Eastes Trust for the relief of registered members of the profession of any age, their widows and orphans, and for the educational assistance of their daughters or sons.

Full information and forms of application are available, on request, from the secretary, Epsom College, Surrey.

Preparations and Appliances

AN "OVERGLOVE" FOR INTRAUTERINE AND OTHER MANIPULATIONS

Dr. J. A. MOYSE, of Mitcham, writes: Access to the uterine cavity, even under the most favourable conditions, is always fraught with the risk of infection. For the practitioner who has to carry out an intrauterine manipulation I have designed what I call the "overglove," a rubber covering for the sterile glove used in cases of adherent placenta requiring manual removal.

The overglove is a retractable covering for the sterile "operating" gloved hand, designed to be carried over it into the potentially septic field of the lower genital tract. At the desired level the overglove is held back by the non-operating hand pulling on the cuff, so that the encased sterile glove can emerge from a suitably shaped slit. The edges of this slit are continued into flaps which are invaginated into the lumen of the overglove and there held between the sides of an appropriate pair of fingers. These flaps are everted as the slit retracts over the emerging sterile gloved hand, which is thus uncovered only in the field of its application. Not having come into contact with anything other than the inner surface of the overglove, the gloved hand must be at that moment in its original degree of asepsis.

FIG. 1

This sequence of movements or manœuvres is shown in the accompanying sketches. Fig. 1 depicts the overglove more or less "in plan" but with the operating hand within it. The non-operating hand, grasping the cuff, is exerting sufficient pull to cause the overglove to retract. The slit is beginning to widen as its flapped edges are everted. Fig. 2 shows the sterile gloved hand emerging from the stretched slit, the flaps of which closely invest it. Continued traction is maintained and the movement completed with the slit retracted to the wrist and the gloved hand fully outside the overglove.

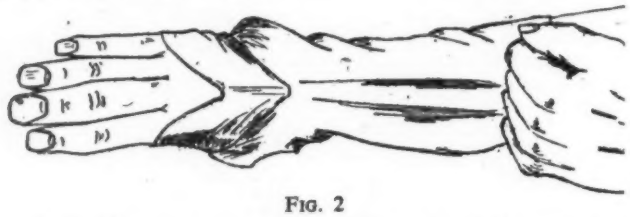

FIG. 2

The overglove is manufactured by Messrs. J. G. Franklin and Sons, Ltd., Colvestone Crescent, Dalston, London, E.8.

Correspondence

Ovarian Hilus Cells

SIR,—While the *Journal* of July 16 (containing our paper entitled "Association of Certain Ovarian Cells with Endometrial Cancer") was being printed, our attention was drawn to a paper by W. H. Sternberg in the *American Journal of Pathology* for May, 1949 (**25**, 493), on the human ovarian hilus cells. The author points out that there is little reference to the cells in the American literature, and we believe that Sternberg's paper is the first full description of the cells written in English. He groups the cells, known to us as paraganglion cells, among the hilus cells, although we feel that the cells we describe belong to a different type. Nevertheless, there is an accurate description of the relation of some of the cells to the blood sinuses and lymphatic spaces. We must emphasize, however, the activity of these cells in cases of carcinoma of the endometrium, particularly in early cases.—We are, etc.,

WILFRED SHAW.
BURJOR DASTUR.

London, W.1.

Tuberculosis in Industry

SIR,—Having been one of those responsible for the report[1] of the Medical Research Committee of 1915 dealing with the incidence of tuberculosis in the boot and shoe industry I am interested in the letters you have published from Dr. Stephen Hall (June 11, p. 1049) and from Dr. S. Roodhouse Gloyne (July 2, p. 31). Both writers, and, indeed, Drs. Alice Stewart and J. P. W. Hughes (May 28, p. 926), seem to have overlooked that the subject received extended statistical study in the Harben Lectures for 1924.[2]

Briefly, the mortality data there quoted indicate that (*a*) occupations with no health risk experience low mortalities from every cause of death; (*b*) occupations "infected" with tuberculosis (e.g., the boot and shoe industry, tailoring, and printing) experience a high mortality from tuberculosis, but low mortalities from every other cause of death; (*c*) occupations exposed to the silica dust hazard experience a high mortality from tuberculosis and from other diseases of the damaged respiratory organs, such as bronchitis and pneumonia, but low mortalities from other causes; and (*d*) occupations exposed to a general depressing agent, such as alcohol (e.g., barman), experience high mortalities from every cause of death.—I am, etc.,

E. L. COLLIS.

Lossiemouth, Moray.

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1 First Report of the Special Committee upon the Incidence of Phthisis in Relation to Occupations, 1915, London.
2 *J. State Med.*, 1925, **33**, 150.

SIR,—I must thank Dr. S. Roodhouse Gloyne for his courteous reply (July 2, p. 31). On rereading my letter (June 11, p. 1049) I can detect some evidence of ill-humour, for which I am sorry. This display of emotion arose because I fancied I saw in the "mistakes" of Drs. Alice Stewart and J. P. W. Hughes symptoms of a habit common among those who hold the bacteriological concept of tuberculosis. They use language which presupposes the truth of their concept, falsely equating "infection" with "disease," and they tend to regard any reproof of this habit as the objection of a pedant. Nevertheless, the offence is rank and fundamental, and its repetition acts as an irritant to those of us who think less highly of the part played by germs in the aetiology of "disease."—I am, etc.,

STEPHEN HALL.

London, W.1.

Incontinentia Pigmenti

SIR,—I read with interest the article by Dr. W. L. Calnan entitled "Congenital Hemiatrophy Associated with Other Congenital Defects" (July 9, p. 56). The history of that case revealed that the child was born with a vesicular eruption on the left leg and dorsum of both hands. There was also linear

pigmentation over the anterior part of the trunk on the left side and over the lateral wall of the thorax. The vesicular eruption left scarring after healing. Furthermore, teeth anomalies developed, and in addition to these defects a congenital hemiatrophy was demonstrable.

The history of a vesicular eruption at birth with peculiar linear pigmentations, combined with other congenital defects, is characteristic of the condition known as incontinentia pigmenti (Bloch–Sulzberger[1]). I had the opportunity of diagnosing three such cases at the Hospital for Sick Children (Skin Dept.—Dr. R. T. Brain). These are going to be published soon. One of the cases was shown at the Dermatological Section of the Royal Society of Medicine by myself,[2] and I feel that Dr. Calnan's case belongs to the aforementioned syndrome and that the hemiatrophy is a new feature which was not described in the previous cases.

Less than twenty cases have so far been described since 1926, but I believe that many cases of incontinentia pigmenti pass undiagnosed.—I am, etc.,

London, W.C.2. H. HABER.

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1 Sulzberger, M. B., *Arch. Derm. Syph., Chicago*, 1938, **38**, 57.
2 *Proc. R. Soc. Med.*, 1948, **41**, 759.

Dysentery in South Persia

SIR,—I have read the article by Dr. Ian S. Stewart (April 16, p. 663) on this subject, and, while the classifications of the different types of bacillary dysentery may be of value, the article generally is not above criticism and I would beg space to make some comments and criticism on it.

In the first place it is hardly accurate to assume that the conditions as found in Abadan may apply to the rest of the province of Khuzistan or those other parts which go to make up the rest of South Persia. There is quite a difference between the conditions of life in, say, Ram Hormuz, a rural native town in the desert plain, and the conditions as found in the industrial area of Abadan lying on the Shatt-al-Arab delta. Secondly, the time interval of the experiment is far too short to justify the conclusions reached that a method of control has been found. Dysentery, like so many other infective diseases, is a seasonal condition and also varies in its intensity from year to year. It would be of interest to know the disease incidence in the previous and succeeding years.

Thirdly, the easy dismissal of amoebiasis, based on the laboratory findings alone, as a condition of importance is not one that many clinicians would make. This condition is notorious for its quiet, unobtrusive, yet sinister presence. Many cases of amoebiasis can only be diagnosed clinically and in their response to anti-amoebic treatment; many cases too seldom have dysenteric symptoms, and *Entamoeba histolytica* may never be found—especially is this so in the chronic form. It is noted also that the use of the sigmoidoscope as an aid to the ascertainment of cure is not mentioned.

My chief complaint is, however, on the assumption by the author that a means of control of bacillary dysentery has been described. This is far from being the case, and, if it is believed that the described method of treating those workers reporting sick with bacillary dysentery "will bring about a steady reduction in the incidence of the dysenteries," it is to be hoped that it is believed by a very small minority.

It is true that this single approach to the eradication of a disease has been the dream of others, as for example the dream that quinine would eradicate malaria, that "606" would eradicate syphilis, or that tryparsamide would eradicate sleeping sickness, but to imagine that the described method will control such an unhygienic condition as dysentery and in such an area as the Persian Gulf, where the sanitation and personal hygiene of the bulk of the people are of a very low standard, is surely wishful thinking at its maximum.

It may come as a surprise to Dr. Stewart and to others to learn that of the 30,000 workers mentioned in the experiment only 15% approximately are housed in anything like reasonable conditions, with modern sanitation, and of this 15% many have still to learn how to use the fittings supplied; most of the remaining 85% live in the congested townships of Abadan town and Ahmadabad, where it is not unusual to find up to six families of three or four persons each living in a single room. Some, indeed, have no living abode at all and sleep on the ground around the covered market-places or in doorways, or share a stable or garage with some animal or car.

While it is true that public lavatories are provided, any vacant space serves as a place where nature's calls may be answered—the local inhabitants not being at all fastidious—and the whole district is mostly polluted by faeces, and flies are legion. I would in all humbleness suggest that if control measures are being applied to control bacillary dysentery those measures suggested in an excellent chapter in that admirable book, *The Control of Common Fevers* (London, 1942), be used; or, where these more modern measures cannot be applied completely, at least the instructions laid down thousands of years ago and described in the book of *Deuteronomy* (xxiii, 13) be carried out. Chemotherapy may have a place in reducing the carrier rate, but nothing can replace the fundamentals of sanitation and personal hygiene in the control of dysentery.—I am, etc.,

Abadan, S. Persia. LAWRENCE G. BRUCE.

Pylorospasm and Uraemia

SIR,—In a study of the toxaemia of acute intestinal obstruction (1923) I used the subtitle "Vomiting as a Pathological Force": the case of pylorospasm reported in the *Journal* (July 2, p. 18) shows how justified was, and is, that opinion. The malaise of sea-sickness, due to an aberration of the blood-flow through the liver occurring before the vomiting begins, is made much worse with the repeated vomiting; and deaths have ensued from sea-sickness alone. The deaths following cholecystectomy, in which the kidneys may become gravely implicated, are to be ascribed to the post-operative vomiting, and only to that. The bad results following caesarean section for eclampsia formerly experienced were due mainly, or indeed solely, to the post-operative effect of the general anaesthesia. With modern methods of rendering the operative area insensitive, by which post-operative vomiting is eliminated (and of which in these cases spinal analgesia is the best), the picture is completely changed; so that we know that in eclampsia the most satisfactory procedure is to empty the uterus, to relieve the vascular disturbance induced by the progressive distension of the abdomen and magnified enormously by the convulsions (in which the abdominal musculature does not become flaccid—the abdomen is as hard as a board) as soon as possible. The day of hypothetical toxins causing pathological changes in all these cases is past.

The case of pylorospasm in an adult related by Dr. E. N. Russell supports this statement. Dr. Russell expresses the opinion that in his case the cure of the gastric trouble resulted from the realization by the patient—a professional man, at the time about 30—that it was due to psychological causes: causes which, I presume, cannot be supposed to have acted by way of producing or of inducing the appearance of a toxin. In treatment, "antal" was peculiarly effective. Antal, we are told, consists of a mixture of atropine and of sodium bromide. Antal cannot be expected to operate by neutralizing a toxin. There was, then, no toxin: yet uraemia occurred. The output of urine varied from 14 to 70 ounces (400 ml.–2 litres) a day: and the blood urea showed considerable excursions—from 200 to 72 mg. per 100 ml. within five days. And the patient, now aged 60, is presumably alive and well. A preceding renal disturbance can be eliminated.

When one considers the structure of the kidney and the long and circuitous course of the tubules through which the urine percolates, it is not difficult to understand that with great contraction of the abdominal wall muscles, such as occurs with vomiting, there is obstruction to the flow of urine and, indeed, to the blood stream within the kidney. The same occurs with strenuous rowing. After a race, the urine of all eight men, each an athlete in perfect health, contained albumin; some of the urines contained blood, and some (I believe) casts (Collier). I have pointed out already that occlusion of the ureter with diuresis in play causes the kidney to become almost bloodless (Brodie); and that a continuous compression of the

kidney itself, such as occurs in concealed accidental haemorrhage, must produce the same result by occluding the Henle hair-pin corners and thrusting bodily the pyramids of the kidney into the renal pelvis, like a cork into a bottle (*British Medical Journal*, 1949, **1**, 584). In the case of repeated vomiting we have a series of compressions : these must present an explanation much more acceptable than the play of any toxin to the student of medicine conversant with the nature and effect of physical force.—I am, etc.,

Rugby. R. H. PARAMORE.

Necropsies and Tuberculous Infection

SIR,—With reference to your annotation (May 21, p. 907) on the above subject, it may be of interest to recall that Hermansson[1] reported as follows in 1939 :

"After examining the carcasses of tuberculous cattle, the meat inspectors washed their hands in sterile water. This was centrifuged, and the sediment injected into guinea-pigs. Eleven of the 15 inoculations produced tuberculosis in the guinea-pigs. . . . The rinse water from the towels used by the meat inspectors was injected into guinea-pigs, producing tuberculosis in every instance."

—I am, etc.,

London, W.C.1. G. P. WEST.

REFERENCE

1 *Skand. vet. tidskr.*, 1939, **29**, 926. Quotation from *Biol. Abstr.*, 1940, **14**, No. 1205.

Large Dose of Curare

SIR,—It is a well-acknowledged fact that patients vary markedly in their susceptibility to different anaesthetics (as indeed to drugs in general). I feel that the case reported below is worth recording, because of the abnormally large amount of curare required by the patient in a relatively short time.

The patient, a young man of 22 in the Merchant Navy, weighing 150 lb. (68 kg.), was to have a partial gastrectomy. There was a history of perforated gastric ulcer at the age of 16 years, with a further operation three weeks later for liver abscess. Unfortunately I have not been able to ascertain the amount of anaesthesia required on these occasions, though obviously curare would not have been used in those days. He was a teetotaller and smoked about ten cigarettes a day.

The anaesthetic technique used has been described elsewhere.[1] Sodium thiopentone (0.75 g.) and *d*-tubocurarine chloride (25 mg.) were required before visual intubation and packing of the throat could be satisfactorily accomplished. Notwithstanding this already significant dose of *d*-tubocurarine chloride, a further 10 mg. had to be given before the abdomen could be explored in comfort. Further maintenance doses were given throughout the operation, the respiration remaining regular and quite deep throughout. Shortly before closure a further dose of 5 mg. *d*-tubocurarine chloride had to be administered, in order to supplement the relaxation for closure of the peritoneum.

The duration of the operation was 90 minutes, during which time a total of 70 mg. of *d*-tubocurarine chloride and 2.5 g. of sodium thiopentone were given. At the termination of the operation "prostigmin" (5 mg.) and atropine (gr. 1/60=1 mg.) were administered, and the patient returned to the ward with good respiratory exchange and in satisfactory general condition. Convalescence was uneventful. All the injections were given through a Gordh needle, which was unquestionably in the vein.

The dose of *d*-tubocurarine chloride in this case was so much greater than any I have experienced or read of that I feel it will be of interest to all anaesthetists.—I am, etc.,

Manchester. M. SWERDLOW.

REFERENCE

1 Chadwick, T. H., and Swerdlow, M., *Anaesthesia*, 1949, **4**, 76.

Treatment of Retained Placenta

SIR,—In these days of synthetic medicine, diplomas, etc., it is refreshing to read some of the facts of life as expressed by those capable of using their powers of observation and confirming them by experience. Mr. W. G. Mills (June 18, p. 1098) puts in a nutshell the true position regarding retained placenta, and Sheehan, from a pathological standpoint, has confirmed that death occurring from this cause is due to shock. Every obstetrician of experience must have repeatedly observed the rapid onset of (often extremely severe) symptoms of shock in patients retaining the placenta with, or equally frequently without, any degree of haemorrhage, even in normal deliveries.

If ever the saying, "He who hesitates is lost," had apt application, retained placenta is the situation bar none. While textbooks still reiterate the two hours' wait after delivery, which must have originated in a generation of mothers very different in their resistance to shock from the present, one can only say from experience how often lives have been saved by making sure the placenta is removed definitely before two hours, and, all things being equal, the longer before the better. In my own domiciliary and hospital practice every midwife long since knows the rule—no placenta inside after one hour.

As to substances necessary for resuscitation (Dr. C. T. H. Whiteside (June 4, p. 1005) and others), in the exceptional cases when this is necessary, if haemorrhage is the cause blood holds the field, but many a life can be saved by either saline or plasma till this can be obtained, or with ancillary restorants.

I cannot agree with Mr. Mills that "the first time the hand is plunged into the uterus in search of the placenta is an awful experience," as that is the very thing which must not occur. Passing of the hand with the utmost gentleness, even over the already stretched perineum and thence through the cervix, is of the highest importance, and failure to recognize this all-important fact is in itself sufficient to precipitate already oncoming shock, even to a fatal issue. While bearing this in mind, it is possible manually to remove a placenta even without anaesthetic in a bad-risk subject, and the writer has more than once been under the necessity of so doing.

While Mr. Mills rightly stresses sterile gown and gloves, he will be fortunate if one night he will not have to be satisfied with thorough scrubbing-up, and hands and arms soaked in antiseptic up to the elbows. In the war years Dodd and others confirmed this as being satisfactory, and more than once I have had to prove its truth, and I have yet to see my first patient to be infected in this way.

I join with him in marvelling at the reluctance of the average practitioner to undertake this simple and relatively easy course out of an often otherwise desperate dilemma, where real terror can only be replaced by sheer joy for all concerned.—I am, etc.,

Altrincham, Cheshire. H. A. LOGAN.

Transfusion in Domiciliary Midwifery

SIR,—As one associated with the obstetrical "flying squads" in the Windsor area I should like to make the following points for consideration. Most emergency calls are made by practitioners because of collapse of their patients of such degree as to make it unwise for them to be transferred to hospital before preliminary resuscitation. The majority of these patients have been delivered of their infants, for cases of ante-partum haemorrhage can usually be sent to hospital by ambulance while their general condition is still satisfactory.

The causes of collapse after childbirth can usually be grouped under the headings : (1) traumatic (? obstetric) shock, without bleeding, and (2) shock following bleeding, with or without associated trauma. With regard to the former—often cases with retained placenta—one might suggest that attempts at manual expression should not be made unless the doctor is prepared to follow this manœuvre with manual removal if necessary. If not, it is better for the patient to be transferred to hospital with the placenta *in utero*, for repeated attempts at manual expression cause profound shock, especially when performed without anaesthetic. Cases of collapse in this group often respond satisfactorily to plasma alone.

If bleeding occurs during the third stage the above principles apply, but uterine massage and intravenous ergometrine can be used in emergency. It is true that the latter may make subsequent removal of the placenta a matter of greater difficulty, but this consideration should not prevent the use of a possibly life-saving measure.

Furthermore, may I make a plea for more frequent blood grouping and rhesus factor estimation during the antenatal period ? In this area these tests are done as a routine in all hospital and council antenatal clinics, and if the patient is given a copy of the report for her retention it may be invaluable in the event of an urgent blood transfusion being necessary at a later date. It is not possible for the "flying squad" to do

more than a simple cross-match in an emergency, and owing to the shortage of rhesus-negative blood, at any rate in this area, the choice may lie between a plasma infusion or the use of rhesus-positive blood, and we have seen some serious complications through the use of the latter. When the rhesus factor is unknown and rhesus-negative blood is not quickly available I would advocate the use of plasma as a temporary measure pending full blood investigation, especially in a multigravida or patient who has had previous blood transfusion.—I am, etc.,

Windsor. F. H. FINLAISON.

Weight-reducing Diet

SIR,—Dr. H. L. Marriott's note (July 2, p. 18) on a causal relationship between the consumption of boiled potatoes and slimming will probably arouse lay interest. Some candidates for slimming, however, might conceivably prefer to follow the advice given to the late Mr. William Banting by the late Mr. Harvey, F.R.C.S., in 1864, and substitute wine and spirits for the drabness of milk and potatoes. The diets published by Mr. Banting and by Dr. Marriott are not greatly dissimilar in other respects.

The respective calorific values of Mr. Banting's daily alcoholic intake and Dr. Marriott's allowance of milk and potatoes are approximately the same, but, whereas a boiled potato is consistently and monotonously just a boiled potato in looks, odour, and taste, there is a wide and interesting selection of wines and spirits with their different vintages, colours, bouquets, palatabilities, and gusts satisfying to the veriest tyro who desires to lose weight pleasantly.

At the ripe age of 66 Mr. Banting lost 46 pounds (20.7 kg.) on his alcohol-positive, milk-and-potato negative diet, and enjoyed doing so. So much interest did Mr. Banting's simple weight-reducing diet (published and distributed free of charge) cause, that some eighty years ago *Punch* broke into verse with a poem that ended thus:

"To avoid, if you wouldn't grow bigger and bigger,
All bread, butter, sugar, milk, taters, and beer.
Take a fresh lease of life and commence a new era
Mr. Banting's advice makes one long to begin—
Drink claret and sherry, good grog and Madeira,
Take four meals a day and—grow gracefully thin."

The passage of almost a century does not appear to have brought about any marked improvement in the art of slimming, and perhaps the old Roman custom (employed to a certain extent in this country around 1824, when Sir Astley Cooper popularized John Hunter's (1772) and Dukes's (1822) modification of what is now Ryle's stomach tube) of eating a hearty meal, pressing on regardless (gastronomically), then vomiting immediately afterwards, had certain advantages.—I am, etc.,

London, W.1. J. DOUGLAS ROBERTSON.

SIR,—May I suggest the use of Dr. R. D. Lawrence's simple diabetic diet (unweighed) card, or a card on the same lines, for the dietetic treatment of obesity? This card, which shows all the common and many of the rarer foods under such headings as "Forbidden" or "Allowed" (more or less *ad lib.*), finally shows a certain number of foods that must be taken in known amounts. A modification of the card gives (*a*) forbidden foods, a group including sugar, cakes, and several other items; (*b*) allowed *ad lib.*—tea, coffee, water, and certain other drinks, saccharin, etc.; (*c*) proteins and fats—here all one need do is say "Normal rations"; (*d*) carbohydrates—(1) a group which may be taken *ad lib.*, (2) a group which should be taken in normal amounts only, and finally (3) there is Dr. Lawrence's last group of several common food items, each called a portion and each being about 10 g. carbohydrate. This last group includes potato (size of hen's egg), bread, milk (a tea-cup), etc. The only item one need weigh (at first) is bread (¾ oz. (18.8 g.) = one portion). If one allows the patient 14 portions per day he (or more often she) makes his (her) selection from this group. By the use of such a diet obese people can join fairly easily in the ordinary family meals. They should weigh themselves weekly, and should aim to lose an average of 2 lb. (0.9 kg.) per week; if not doing so after two or three weeks they can raise or reduce their number of portions slightly. It has been suggested that one item—nuts—not specially excluded on the diabetic diet card should be excluded in the treatment of obesity, but in general their cost will forbid them to most people.

The card is obtained from Mesrs. H. K. Lewis & Co., London.

In using Dr. Lawrence's diabetic card one must of course delete such paragraphs as relate peculiarly to diabetics—e.g., reference to insulin.—I am, etc.,

Middlesbrough. BASIL LONGBOTHAM.

SIR,—Dr. H. L. Marriott's weight-reducing diet (July 2, p. 18) allows as many potatoes as the patient likes, but no fat or cheese. A variation of this diet which is equally successful and pleasanter for the patient is to forbid potatoes but insist that all the ration of butter, margarine, cooking fat, and bacon is eaten, as well as all the cheese which can be procured. Patients should also be told they must eat four meals or snacks daily, and if they find that as a result of the diet they are sleeping badly or are having nightmares they should have their largest meal as late as possible at night.—I am, etc.,

London, W.1. FRANKLIN BICKNELL.

Teaching Pharmacology in India

SIR,—I was much impressed with the report of the Medical Curriculum Committee on "The Training of a Doctor." Especially was I pleased to read the remarks on the teaching of pharmacology and therapeutics.

Having devoted now over 30 years to the teaching of pharmacology, and being the author of a standard book on pharmacology, materia medica, and therapeutics, I always realized the defect in the present system, and you were good enough to publish in the *Journal* of Sept. 11, 1937 (p. 552), my views on the teaching of pharmacology. I stressed the importance of teaching pharmacology and therapeutics together and also the necessity of allotting clinical beds to the teacher of pharmacology.

Recently the Medical Council of India appointed a committee to revise the medical curriculum, and I have been appointed chairman of the Committee on Pharmacology and Therapeutics. Your report has been of great help to us. I have issued a questionary to get the viewpoints of those who are engaged or interested in the subject. I shall be grateful if you could help me in getting the views of those interested in the subject in your country.—I am, etc.,

Pharmacological Department,
R. G. Kar Medical College,
1, Belgachia Road, Calcutta. B. N. GHOSH.

Tolerance to Adrenaline

SIR,—The following case illustrates acquisition of a very high degree of tolerance and resistance to adrenaline, followed by complete loss of same after the drug had been withheld for a short period.

The patient, a man aged 51, has had spasmodic asthma for four years. He suffers from recurrent bouts of what can best be described as chronic status asthmaticus. These bouts last about a month. Before coming under my care he was taught how to administer subcutaneous doses of adrenaline to himself. Though restricted as to what was a safe dosage, he was urged by the severity of his symptoms to adopt "kill or cure" methods. On one occasion he injected 180 minims (10.5 ml.) of adrenaline hydrochloride solution (1/1,000) as a single dose. He remembers no subjective symptoms. When the patient came under my care he was using large quantities of the drug in divided doses—i.e., 3–4 minims (0.18–0.24 ml.)—every minute until relief of symptoms, perhaps 1 oz. (28.4 ml.) in all during a bad night. The injections sometimes brought on an occipital headache.

After the last prolonged bout came to an end the drug was withheld for a month, and during this time there occurred a remarkable loss of tolerance. When next I had occasion to use the drug I was agreeably surprised to find that a 15-minim (0.9-ml.) dose was effective. This caused the systolic blood pressure to rise from 125 to 175 mm. Hg in six minutes, together with a small rise in the diastolic pressure and the pulse rate. The next day a larger amount was necessary to relieve his asthma. It caused a most violent reaction (agonizing throbbing headache, rigor, and tachycardia), and even a 15-minim dose on the following morning caused a similar headache.

Thus a patient who had once taken a single dose of 180 minims of adrenaline hydrochloride (1/1,000) without subjective ill effects became intolerant to a dose of 15 minims when the drug was withheld for a month. There was no question of the needle being in a vein.—I am, etc.,

Dublin. E. DESMOND FLOOD.

Exchange of Doctors with U.S.A.

SIR,—Before the war there were some schemes in action for the exchange of doctors between hospitals in this country and the U.S.A. There is now a more widespread desire by doctors in this country to visit the U.S.A. This may often be facilitated by arranging for a regular exchange of doctors between medical schools and hospitals in the two countries. There seems to be some fear that making such an arrangement for an American doctor to work in a hospital here infringes the Acts governing medical practice here. This is not the case, for by Section 8 of the Medical Practitioners and Pharmacists Act, 1947, Parliament has made provision for the admission to the *Medical Register* of Commonwealth and foreign practitioners temporarily in the employ of hospitals in this country.

If a hospital is prepared to offer a postgraduate post to a qualified practitioner who is not registered here, it is desirable both for him and for the hospital that he should have the status of a registered medical practitioner as the holder of that post, and Section 8 enables him to become temporarily registered here for that purpose.

The procedure for temporary registration under the section is quite simple, and it is only necessary to add that a practitioner who is not a British subject must, of course, as a first step obtain permission from the Home Office to enter and to remain in the U.K. in order to do postgraduate work.—I am, etc.,

London, S.E.1. RONALD MACKEITH.

POINTS FROM LETTERS

Palpation of the Pancreas

Dr. N. S. FRASER (St. John's, Newfoundland) writes: Is it possible to palpate the pancreas in disease? The normal pancreas is soft and could not possibly be felt through the abdominal wall, but a diseased gland, swollen and congested, will be firm to the touch and has only a flaccid, empty stomach over it. The transverse colon does not cover it. Then there is the abdominal wall with its tense rectus abdominis which is liable to contract if the hand is not gently applied. The inscriptiones tendinae are above and below the area to be palpated, and so do not interfere with the palpation. With concentrated attention, a warm hand flat on the abdomen, and light pressure, gradually increased, one can feel the whole gland if it is swollen with disease. Then, on pressure with one finger, the patient will wince and say it hurts. Such has been my finding in the diagnosis of subacute pancreatitis, beginning with a personal experience, and I would like an expression of opinion from others. The spleen cannot be felt until it is diseased, and the same can be said of the kidney; so why not the pancreas?

Herpes, Varicella, and Liver Extract

Dr. L. KILROE (Rochdale) writes: With reference to the letters by Drs. James Rooth and M. Mansour (July 9, p. 99), I saw cases of herpes coinciding with attacks of chicken-pox in 1940 and 1946. Both occurred in adult males (aged 45 and 47). The herpes, in one case intercostal and in the other frontal, appeared 5–7 days before the chicken-pox rash. Incidentally, the injection of 2 ml. of "anahaemin" in the second case on the appearance of the herpes did not appear to modify the course of either the herpes or the chicken-pox. The eruption in the intercostal case was the worst I have seen in a case of shingles. In over 40 years of practice I can only recall one other case of severe chicken-pox in an adult, again a male, aged 52. Herpes was not present. I have not yet seen a case of herpes with chicken-pox in a child.

Creeping Eruption

Dr. F. ADAM THOMSON (Ipoh, Malaya) writes: With reference to the medical memorandum by Dr. J. P. Caplan (March 5, p. 396), a number of these cases came under my care while practising in Northern Rhodesia. Infection as a rule seemed to date from a South African holiday. All responded to the vigorous local application of oil of chenopodium to the advancing end of the weal.

Obituary

BRIAN RAIT-SMITH, D.A., F.F.A. R.C.S.

Dr. Brian Rait-Smith, anaesthetist and demonstrator of anaesthetics to St. Bartholomew's Hospital, died at St. Albans on July 14, aged 45. He was educated at Jesus College, Cambridge, and St. Bartholomew's Hospital. He qualified in 1929. Within six months of entering the department of anaesthetics as a junior he was promoted to senior resident anaesthetist, an appointment he held until he joined the honorary staff in 1937. He took the D.A. in 1935, and this year was admitted F.F.A. R.C.S. He was particularly interested in the anaesthesia of neurosurgery and thoracic surgery. As a resident he played an important part in the pioneer work of chest surgery, and until his death gave anaesthetics for the increasingly difficult procedures undertaken by thoracic surgeons and neurosurgeons. He published work on anaesthesia for cardiac surgery, particularly that of the patent ductus arteriosus, and his article on anaesthesia in neurosurgery is shortly to appear in a textbook. He also wrote on obstetric anaesthesia, and his own observations on trichlorethylene and curare were reflected in papers from his department. Before he became whole-time anaesthetist to St. Bartholomew's at Hill End Hospital he was anaesthetist to the British Postgraduate Medical School, the Brompton Hospital, and Papworth Settlement, and he had been consulting anaesthetist to the L.C.C.

To the surgeons with whom he worked and his colleagues in the department Rait-Smith's skill was a greatly appreciated constant among the many variables of operative surgery. The difficult work he undertook is proof of the high standard of his anaesthesia. He had all the properties of a first-rate anaesthetist—ability, cool-headedness, sharp clinical judgment, and an enviable amount of common sense. That he was always cheerful and courteous and never allowed his temper to become ruffled was remarkable in one who spent his life in busy operating theatres. His residents became his firm friends within a few weeks of their appointment, and his help in their later careers can be witnessed to-day by a score of successful anaesthetists. A hard worker, he never resented a call to the theatres at Barts, however inconvenient the hour the request was made. There was no "side" about Rait-Smith. He hated affectation, and was as skilful in his deflation of a self-important surgeon as in his discreet encouragement of a young surgeon or anaesthetist. All this he managed despite the subarachnoid haemorrhage he had in 1942, the significance of which he knew well, and two major operations in the following five years. He was so full of zest for life in spite of his ill-health that his death is a shock to his friends, who will all extend their sympathy to his wife.

A memorial service will be held in the church of Saint Bartholomew-the-Less, on Tuesday, July 26, at 12.30 p.m.

Dr. CHARLES SALKELD, who died in Bournemouth on June 7 at the age of 81, was for many years in practice as an ear-nose-and-throat specialist in that town. He was educated at Manchester Grammar School and served his time as an engineer before taking up medicine. After graduating B.A. with honours in classics in 1891, he had a brilliant career at Durham University. He won the Gibb Scholarship in pathology, the Dickinson Scholarship, and the Charlton Scholarship in medicine, all in 1896. He graduated M.B., B.S. a year later. After 12 years

of general practice in Leeds Dr. Salkeld decided to specialize in ear, nose, and throat work, and he studied at the Central London Throat and Ear Hospital, where he became a clinical assistant. He started specialist practice in Bournemouth in 1913, but in 1915 joined the R.A.M.C., in which he served at home and overseas for four years. He then returned to Bournemouth, where he was appointed to the staff of the Cornelia and East Dorset Hospital, Poole, and later the Cottage and Children's Hospitals at Swanage. He also operated for many years at the Victoria Home for Crippled Children. Dr. Salkeld had recently retired from active practice following a severe illness. He had had an unusually broad education in engineering, classics, and medicine. His interests were wide, and his range of accurate knowledge was a byword with his family and friends. Apart from all this Charles Salkeld had a personality and appearance which inspired confidence and respect. He had a quiet, cheerful disposition, and when he spoke it was usually to give a well-considered opinion. Small talk and making conversation were not in his line; and he disliked social functions, at which he seldom appeared. He was unostentatious, never pompous, and completely genuine. His loss will be keenly felt by many friends, relatives, and colleagues. He leaves one son, who is in practice as an ear-nose-and-throat specialist in Bournemouth.

Medico-Legal

LEGACIES TO HOSPITALS UNDER N.H.S.

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

A lady made her will on June 18, 1948, directing the executors to divide the residue of her estate into eight equal parts and to give one part to the Cancer Research Institute and another to the Royal Cancer Hospital.[1] At that time, and when she died on June 23, 1948, both were charities, but before the will was proved the National Health Service Act came into force, on July 5, 1948, and doubt arose whether the right to receive the share of the residue left to the Institute was vested in the Royal Cancer Hospital or in the Minister of Health. Was the executor required to apply the shares to the board of governors of this teaching hospital under Section 60 of the Act, or to the Ministry, or were they undisposed of? These questions were brought to the Chancery Division for solution by the Ministry of Health, counsel admitting that the Ministry confessed itself unable to understand the Act. The Ministry desired elucidation of the Act for its own convenience.[2]

Mr. Justice Harman said in his judgment that he had expected to find some guidance in the Act on the destination of legacies to hospitals. He had read the Act section by section with a bewilderment which finally ripened into despair when the conclusion was forced upon him—a conclusion from which he could see no escape—that by some mischance the Legislature had so framed the clauses of the Act as to leave charitable legacies of this kind outside it. The Act in that respect was, he said, almost a miracle of ineptitude.

The question could not go to arbitration, for it was a matter of law and not of fact. The interest which was left by the will was excluded from the category of endowments, nor was it a right acquired for the purpose of carrying on the business of the hospital. As the legatees had survived the testatrix, the legacies could not have lapsed.

The bequest must be dealt with by a scheme brought into court by the Attorney-General in the ordinary way. In discussing costs, he said that the difficulty had been caused by the astonishing ineptitude with which Part II of the Act was drawn, and he saw no reason why the charities which took the other shares of the residue should pay for the omissions of the Legislature. He did not think he ought to order the Minister to pay the costs, but he ordered that they should come out of the two shares over which the difficulty had arisen.

1 *The Times*, May 12, 1949
2 *Manchester Guardian*, May 12, 1949.

Medical Notes in Parliament

TUBERCULOSIS IN SCOTLAND

Estimates for the Department of Health for Scotland and the National Health Service, Scotland, were considered on July 6 in the Scottish Standing Committee.

Opening the debate, Mr. WALTER ELLIOT drew attention to recent figures for the incidence of pulmonary tuberculosis in Scotland. The factual inquiry which the Secretary of State for Scotland had set on foot had not yet indicated the nature of its report. Would it be available before the House rose for the summer?

Mr. WOODBURN said the report would not be available. Many facts had been elucidated, but if they were to be published as they stood at present they would be misleading and in some cases alarming.

Mr. ELLIOT said that in the absence of information exaggerated figures held the field. He hoped it would be possible to bring out an interim report and that some parallel figures could be secured for England and Wales. Figures which he himself had given to the Committee last year had indicated that the good figures for tuberculosis were in the South of England and that as one moved to the North the figures increased. This was not conclusive, however, because when one came to Aberdeen the figures suddenly improved. Dr. Harry Rae, the medical officer of health for Aberdeen, had pointed out in his report for 1947 that, although in Scotland as a whole the death rate for pulmonary tuberculosis was 66 per 100,000, in Aberdeen it was 35. In Scotland generally there had been increases both in the number of deaths and in notifications. The number of deaths was 2,717 in 1939; 2,932 in 1945; 3,231 in 1946; 3,739 in 1947; and 3,417 in 1948. Notifications were 7,317 in 1945; 7,627 in 1946; 7,984 in 1947; and 8,010 in 1948, which was the highest figure in Scotland for 30 years.

The figures for non-pulmonary tuberculosis were improving. That reflected success in the long campaign against bovine tuberculosis. In 1945 they were 18, which was higher than pre-war; 15 in 1946; 14 in 1947; and in 1948 they were down to 9. In Scotland the number of patients in institutions had risen from 4,253 in 1945 to 4,462 in 1948. The waiting-list had gone up from 1,600 in 1945 to 2,500 to-day. Yet there were still in Scotland 572 beds which because of lack of staff could not be used. Dr. Harry Rae attributed part of the favourable position of Aberdeen to the fact that there were enough beds available there.

At the recent Harrogate conference a Glasgow doctor had spoken of a large increase in the waiting-list there and drew attention to an increase in the proportion of notifications among children under 15—in 1945 it was 9.3%, but in 1948 it was 18.4%. Glasgow used to have its own tuberculosis organization. Now the responsibility had moved to the Glasgow Regional Board. The National Association for the Prevention of Tuberculosis had advised regional hospital boards to set up a permanent tuberculosis committee. He did not know to what extent that advice had been carried out in Scotland.

A more extended use of B.C.G. vaccine might be of advantage and might help to stop the wastage of nurses in tuberculosis institutions, which rose as high as 65%. But isolation must always remain one of the main weapons for dealing with the white scourge. The number of active cases of tuberculosis on the waiting-list for houses in Glasgow was 437 in 1945 and in 1948 was 593. The Secretary of State must regard this as a matter of urgency. In some way or another the 500 beds which were empty in sanatoria should be brought into use. Vigorous campaigning by the Secretary of State would obtain an increase in the number of nurses for sanatoria.

Some post-war deficiency in nutrition was perhaps responsible for part of the increase. The intake of calories per head was less than it had been before the war. People in Glasgow, although badly housed, had before the war spent more money on food. Now they could not get more food than anyone else. It might be that somehow a more active tubercle infection had

reached the country. The great influx from the Continent, the influx of Poles, might have something to do with that.

Changes in Incidence

Mr. WOODBURN said the main increases had taken place in districts where the Poles had not been. The incidence of tuberculosis was very high in Southern Ireland, but there never had been a smaller importation from Southern Ireland than in the period when tuberculosis in Scotland had increased. The fall in deaths from non-pulmonary tuberculosis was from 705 to 426. Notifications of tuberculosis had gone up by a third compared with the five years before the war. But a comparison between notifications to-day and pre-war notifications had no value except to show that the medical profession was more alert to deal with the incipient disease. The number of notifications gave no indication of the reason for the increase in deaths.

Investigations by the Committee which he had set up had revealed how difficult it was to reach any sound conclusions. Deaths from pulmonary tuberculosis had been rising in Scotland but not in every place. The picture Mr. Elliot had given of children under 15 was not quite accurate; actually the number of cases had decreased slightly even when compared with the pre-war figures.

The situation could not be explained by any figures available at the present time. Because of this he thought it would be unwise to publish figures which might create speculation and alarm. In the worst places in Scotland the incidence was 110 per 100,000, and in the best places a little over 10 per 100,000. Congested living conditions still had a great deal to do with the spread of the disease, and therefore they should concentrate on segregation. So far as any distinction could be drawn it was between congested old towns and country areas, yet Ross and Cromarty was a bad area.

He had taken steps to get an additional 1,000 aluminium houses so as to free houses suitable for families where tuberculosis was present. The Department had also been able to make an additional 500 beds available since the beginning of 1948. It had put 50 more full-time nurses and 350 part-time nurses on to this work. Streptomycin was available in all cases where it was suitable for use. B.C.G. would shortly be available for use in Scotland for contacts. It was true that Scotland was still going backwards in regard to tuberculosis, and that tendency had not altered this year. The mystery was that they were not going backward in every place. The problem had been narrowed down, and the Committee under the National Health Services Council was pursuing this matter vigorously.

Mrs. CULLEN suggested that Mr. Woodburn should consider moving elsewhere aged and infirm persons who did not require trained nursing personnel. This would clear some wards so that the trained nurses could be used to greater advantage.

Commander HUTCHISON said a more extended use might be made of health visitors to keep cases of tuberculosis under observation. There were four mass radiography units in Scotland, and it would be helpful if there were more.

Mr. HUBBARD urged that a campaign should be started by the Government giving information to every individual about the causes of tuberculosis and the means of preventing its spread.

Position of Tuberculosis Officers

Mr. GALBRAITH urged Mr. Woodburn to ask the Committee to issue some sort of report at the earliest possible moment. He was doubtful whether patients would continue to get the best possible treatment if tuberculosis officers were classified as senior hospital officers and not as specialists. This fear had been expressed at the B.M.A. meeting at Harrogate. They needed to attract the most able doctors to the study of tuberculosis.

Mr. MATHERS said doctors with whom he had discussed the rise in the incidence of tuberculosis had suggested that the conditions of life of young people, and particularly young women, were important. In his constituency young people left home early in the morning to go to work by bus, had hurried meals, and were out dancing till midnight three, four, or five nights a week. As a result they became debilitated and an easy prey to tuberculosis. A great deal of the incidence could be prevented by instruction to people about the way they sapped their energy and reduced their resistance. The highest incidence of tuberculosis was between the ages of 15 and 25.

Replying to the debate, Mr. THOMAS FRASER said Mr. Woodburn was anxious that as much information as possible should be made available to health authorities and Members of Parliament as well as to the medical profession. The Committee which had been set up must conduct its researches thoroughly before reporting. It might be that there was a deficiency in diet, and the Committee would consider that. The diet of Scotland, however, was no different from that of England and Wales. The number of notifications in children under 15 had gone up a little, perhaps because of mass radiography, but the death rate of those under 15 was going down. They must not imagine that the position was improving in the whole of England and Wales. The town where figures had worsened most in the last 10 years was Jarrow. In 1938 Jarrow had 78 deaths from tuberculosis per 100,000 population. In 1947 the figure was 110. Glasgow was second in the list with 85 deaths in 1938 and 105 in 1947. Next came Dundee with 62 deaths per 100,000 in 1938 and 82 in 1947. The worsening was where poverty had been greatest in the last 20 years.

Concluding, he pointed out that the general health of Scotland was better than it ever had been before. The suggestion made by Mrs. Cullen that nurses now looking after old people should be made available for cases of tuberculosis was well worth making and had not been overlooked by the Secretary of State or by the hospital boards.

The Committee then adjourned.

General Medical Council

Sir HARTLEY SHAWCROSS moved on July 12 the Second Reading of the House of Commons (Indemnification of Certain Members) Bill. He explained that it dealt with a possible infringement of the laws relating to offices of profit under the Crown. These laws in the main were embodied in the Succession to the Crown Act of 1707 and were obscure. The view had been taken that to be held under the Crown an office did not necessarily have to be subject to any continuing control by the Crown in its exercise. Where there was any expectation of payment going beyond a fair reimbursement of expenses incurred, such payment would make the office one of profit. The attention of the Government had been drawn to the position of certain members who had been nominated by his Majesty as members of the General Medical Council. The King, on the advice of the Privy Council, nominated five members of the Council to hold office for a term not exceeding five years. Provision was made for the payment of fees to members, and in practice they were paid a sum of 5 guineas in respect of each attendance. They were not paid out of moneys provided by Parliament but out of moneys collected by the medical profession from registration fees. That the actual emoluments of an office were derived from sources other than public funds was not held by the Government to be a criterion of whether an office was one of profit under the Crown. During the present Parliament two Members of the House had been nominated to the Council on the advice of the Privy Council. Previously many members had sat on the General Medical Council without anyone suggesting that there might be an infringement of the Statute of 1707. Penalties against these members should now be statute barred and they were not included in the Bill.

The two members appointed in the lifetime of the present Parliament had been appointed and held their office in good faith on the basis of many precedents, but there was no doubt that they had been appointed to an office of profit under the Crown. Mr. John Hynd had never acted in the office, because before he took any part in the proceedings of the General Medical Council it occurred to him that there might be some question of impropriety in holding the office. The two Members concerned must vacate the appointments but ought not to be penalized for having held them, and this Bill sought to indemnify them. The Government did not seek to constitute the office into one not of profit, and did not propose to legislate to say this should not be an office which members must not hold. It would be the future policy that no member should be

qualified to hold the office. Sir Hartley went on to refer to a case of a different character in which another member was concerned and for which the Bill also proposed indemnification.

Mr. R. A. BUTLER said that, in view of the fact that under the rules of the General Medical Council fees and reasonable travelling expenses could be paid, there was no doubt that the case of the two members ought to be covered by a Bill such as that before the House. It was intolerable that members should be in a position where they might at any time unwittingly contravene the law.

The Bill was then read a second time without a division, passed through Committee without amendment, reported, and read a third time.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

Sir Maurice Alan Cassidy, Physician-in-Ordinary to the King, has been elected an Honorary Fellow of Clare College

David Robertson Smith, M.B., B.Chir., has been elected to an Elmore Medical Research Studentship, from May 1.

In Congregation on June 21 the degree of M.D. was conferred on T. S. Hindle.

In Congregation on June 22 the degree of M.B. was conferred, by proxy, on J. Ff. Edwards.

UNIVERSITY OF GLASGOW

The following medical degrees were conferred on July 9:

M.D.—A. Allison, W. Chambers, W. W. Douglas, W. S. Linton, T. M. Paterson.

Ch.M.—J. Cuthbert, A. W. Kay.

M.B., Ch.B.—Kathleen M. Adams, Marion K. Aitken, A. G. Anderson, G. Anderson, I. N. O. Asinobi, H. B. M. Auld, Anne R. Austin, A. G. Baikie, M. McA. Baird, R. Bankier, G. I. Barrow, N. J. B. Bowie, A. H. Boyd, A. Brodie, I. D. Brown, T. Brown, J. Buchanan, Millicent E. Burns, R. K. Burt, A. H. P. Carswell, Mary S. Christian, J. H Cowie, I. D. Cullen, T. A. Cunningham, D. Davies, F. W. G. Deighton, D. A. T. Dick, J. F. Dick, Grace C. Dunbar, R. Erskine, J. Forsyth, J. Frame, P. G. Gaskell, R. M. Gibson, J. C. Gillespie, J. S. Gillespie, B. Green, R. Greenhill, A. J. Hale, A. M. Halliday, P. Hay, J. C. M. Henderson, G. P. Henry, W. F. Heslin, Elspeth C. Higgins, Margaret I. Hill, D. S. Hogg, D. W. G. Honeyman, R. McD. Hosie, Janet L. P. Hunter, S. W. Hind, Christina M. Jack, J. B. Johnson, D. G. Kay, Mary W. Kellock, A. E. D. Kennedy, A. S. L. Kennedy, J. Knox, A. Kyle, A. S. McC. Lamont, J. G. Leitch, S. G. Leitch, D. W. Leslie, G. H. Lowther, S. G. McAlpine, Sheena E. McCoshim, D. MacDonald, D. G. MacDonald, Sheila C. MacGregor, N. J. McKellar, J. V. McKenzie, Ruth B. McKenzie, D. McKirdy, J. R. Maclean, Ethel E. M. McLellan, Cynthia J. McPherson, W. McQuater, R. K. McRae, G. T. Mair, Maureen A. F. Malcolm, Hazel G. Mansell, Elizabeth L. Millar, T. M. Miller, A. T. Mitchell, J. N. S. Mitchell, J. J. E. D. Mitchell, J. Moodie, J. B. Morris, Sylvia Morris, T. P. O'Hea, H. W. J. Park, L. R. Parker, D. C. Pendrigh, A. J. E. Pollock, W. M. Prentice, J. R. Raeside, A. M. Reid, A. W. Reid, J. M. Reid, B. J. Reilly, A. A. Robertson, A. K. Ross, P. Roughead, J. R. Roy, T. S. Russell, T. C. Rutledge, Jessie L. Sharp, A. Silver, R. G. Sinclair, G. D. Smellie, C. McP. Smith, Joan E. Smith, W. D. Smith, Sheila W. Soote, R. H. Stewart, W. O. Stewart, D. S. Strachan, G. H. Swanson, Vera Taylor, Elizabeth D. M. J. Thomas, A. S. Watson, Sheila M. Whiteside, J. R. M. Wilson, R. B. Wilson, W. Wilson, G. B. Wylie, G L. Wylie, Mary W. Young.

UNIVERSITY OF DUBLIN

SCHOOL OF PHYSIC, TRINITY COLLEGE

The following candidates have been approved at the examinations indicated:

M.D.—R. A. J. Holmes-Ievers, P. J. Mullaney.

M.B., B.Ch., B.A.O.—D. W. Beckett, W. G. T. Bell, F. G. O. Burrows, Dorothy J. Cartmill, C. G. H. Charlton, R. P. A. Coffey, R. S. Crone, W. P. C. Deely, E. W. Eppel, B. B. Fazackerley, T. G. Feeney, [1] [2]Audrey M. Franklin, [1]R. J. M. Fry, [2]Agnes C. Good, C. Harris, Wilhelmina Hutchinson, H. P. Isaacson, [1]Beatrice P. Karmel, S. M. Laird, [1]Thelma H. W. Morks, Sybil E. K. Oulton, [1] [2]Eleanor B. Outon, W. Pappenheim, L. R. Robinson, Kathleen C. Rogers, Eva Roith, C. F. Ross, [1]Stephanie Saville, [1]S. B. Sloan, M. D. M. Staunton, B. S. Telford, [2]W. D. H. Troughton, E. R. D. Williamson, Oonagh D. Wray, R. S Young.

M.B.—Reva Berstock, S. C. Blake, Evelyn Browne, Joan I. Buchanan, L. J. Clein, K. C. Finkel, R. C. Gray, O. H. Killen, E. B. McHugh, Sheilagh M. McHugh, J. C. McLean, W. D. McNaul, T. T. E. Michael, R. Nelson, J. P. Nixon, V. P. O'Sullivan-Quinn, Ruth M. Perrott, Frances E. Russell, R. N. Siberry, A. A. Sweeney, Annie M. G. Troughton, F. A. Walkey.

M.B., B.A.O.—Vyvyan I. K. Johnston.

B.Ch., B.A.O.—Mabel M. H. Hodgins, [2]B. A. McC. Thompson.

B.Ch.—W. D. H. Powell, Gwendoline G. L. Poots.

B.A.O.—D. L. Cowan, Marisa A. O'Keeffe.

[1]With second-class honours in medicine. [2]With second-class honours in surgery.

UNIVERSITY OF DURHAM

In Congregations held on July 1 and 2 the following medical degrees were conferred:

M.D.—H. M. Dixon, D. G. French, S. Josephs, F. J. Prime, M. Walton, *C. A. H. Watts, *W. E. Hick, *C. Strang.

M.S.—James Watt.

M.B., B.S.—C. F. Anderson, J. A. Anderson, G. W. Bainbridge, Jean M. Bainbridge, S. M. Bell, G. S. Black, Sheila M. Bowerbank, F. W. B. Breakey, Hugh Brown, Eleanor J. Carmichael, Barbara W. Cooke, L. D. J. Coore, D. Coulthard, B. W. D. Crawford, Elizabeth Daley, H. Davison, H. D. Davison, Mary Dixon, Joyce Dodds, L. B. Fleming, Robina M. Forsyth, Vera M. Gallagher, Dorothy B. Gilmour, J. B. Glaholm, Pearl Goddard, H. Gossmann, Eve R. Halson, J. Harper, Maureen McG. Henderson, J. P. Hurley, J. E. M. Hutchinson, W. D. Kerr, E. G. Knox, G. E. Langley, Sheila Langley, L. J. Levene, Norma D. Lill, J. McCormack, J. R. McGregor, I. F. McNeill, Joan E. Maddison, J. S. Makepeace, R. W. Mayes, Ann Moffatt, Sylvia A. Moss, G. A. Nemeth, Muriel K. Nicol, P. G. F. Nixon, Ruth Parkinson, Dorothy A. Percy, A. B. Piper, Ann F. Pumphrey, Greta L. Reid, W. T. Richardson, Margaret W. Rodham, N. R. Rowell, C. M. Scott, Gillian E. Sladden, A. S. Todd, C. G. Tulloh, H. P. Tulloh, J. Turnbull, W. Turnbull, Constance N. Vernier, Elizabeth Wales, E. W. Walton, A. J. Watson, L. L. Williams, R. K. Epstein, *G. H. N. Bates, *J. M. H. Hopper, *Kathleen S. Reed, *Winifred J. Skinner.

**In absentia.*

UNIVERSITY OF LONDON

The following candidates at the London School of Hygiene and Tropical Medicine have been approved at the examination indicated:

ACADEMIC POSTGRADUATE DIPLOMA IN PUBLIC HEALTH.—A. O. Abushamma, René F. Barclay, S. Y. Bhagwat, *H. Binysh, J. F. E. Bloss, L. B. De Lanerolle, H. A. Dharamsey, J. E. G. Earle, L. G. Eddey, T. P. Eddy, S. J. Evans, A. F. Fowler, *A. W. Gilks, A. H. Griffith, R. F. Guymer, W. G. Harding, C. H. Hoskyn, Esther Jackson, S. A. Jones, W. A. Karunaratne, M. S. Kataria, D. A. Kuruppu, M. G. Langdon, P. A. Lee, R. N. C. McCurdy, J. C. McDonald, T. C. Macdonald, J. J. McNair, J. R. Mayers, Isobel S. Mowat, G. M. O'Donnell, C. E. Phillip, Isabella M. Pinkerton, J. St. C. Polson, R. H. Pratt, P. G. Roads, C. L. E. H. Sharp, R. A. G. Smith, D. G. Snell, W. B. Thorburn, R. N. Wilson.

*Awarded a mark of distinction.

UNIVERSITY OF MANCHESTER

The following candidates have been approved at the examinations indicated:

M.D.—[1]J. Curry, B. H. Dawson, [2]T. B. S. Dick, [1]F. Howarth, [2]W. E. Kershaw, F. Latham, F. S. Mooney, [2]P. H. Renton, [2]E. P. Samuel, D. Sutton.

Ch.M.—R. H. Sewell.

FINAL M.B., Ch.B.—[3] [4]E. A. Allcock, [3] [4]Jean A. Lovatt, [3]W. J. Metcalfe, Mary A. Ad'lington, Anne R. Barlow, [4]Jessie M. Beard, T. B. Benson, K. Bloor, Margery F. Blumberg, F. Bottomley, Jean M. Breakell, N. C. Brown, K. D. Buckley, P. M. L. Burne, F. R. Calvert, [4]Betty Carruthers, C. Cohen, P. Cuff, R. D. Currie, S. Dobson, Audrey E. Draycott, P. D. Drinkwater, [4] [6]J. B. Eagles, G. Fildes, Edith H. Grundy, G. Hudson, A. G. Jackson, G. Kettlewell, Kathleen M. Lord, I. Macpherson, Lavinia M. Mallott, Marguerite Midgley, Barbara Oldham, F. W. Oliver, Christine M. Parish, Marjorie E. Petch, N. E. Quin, Regina Reif, Marjorie Rivers, A. D. Robinson, M. Sandler, Jeanette S. Shankland, D. P. Shepherd, Rosaleen E. Singer, I. A. Susman, R. M. Thomson, T. A. J. Thorp, Margaret R. Thoseby, Barbara J. Walker, Kathleen R. Walker, T. H. Whitaker, Olive C. Wilks, D. H. Yates. *Part I:* B. L. Alexander, D. Ashworth, R. D. Atherton, Constance M.

Bevan, G. Bills, Flora M. Bisset-Smith, S. M. Broman, P. Burgess, J. C. Canney, P. W. Carnie, Muriel D. Carr, G. Clarke, Barbara M. Couldwell, D. Davies, B. Didsbury, J. Ellis, E. J. Epstein, J. D. Evans, M. C. Flasher, M. Fox, G. Garrett, G. R. Gates, F. G. Hardman, J. K. Harris, T. V. A. Harry, B. Higgins, R. Hinchcliffe, J. T. W. Howe, G. Howitt, T. R. Hunter, J. E. Kench, W. Kerns, P. B. Lees, B. J. Leonard, M. A. Lerner, P. F. Lister, W. H. Lyle, Maria R. Mackay, B. E. Marks, A. W. Moule, J. T. Newton, A. S. Nicholas, G. W. H. Oldfield, Nancy Parkin, D. W. Preston, R. Price, G. V. Purnell, K. L. Purnell, M. B. Raby, Carolyn C. Rigby, Gertrude H. G. Robinson, J. Robinson, Dora M. J. Rosbotham, D. S. Rosenberg, Kathleen M. Rushton, R. Schofield, H. Selcon, Lorna J. Shankland, S. Shubert, A. Sippert, D. M. Somerville, S. Stead, G. Sullivan, Hazel W. Symon, C. F. H. Vickers, Margaret R. Walley, G. M. Watson, Sheila M. E. West, W. A. Whittaker, B. A. Wolfenden, D. Wolman, A. M. Womack.

DIPLOMA IN PSYCHOLOGICAL MEDICINE.—*Part I:* M. M. Lidgate, A. Model.

[1]Gold medal. [2]With commendation. [3]Second-class honours. [4]Distinction in medicine. [5]Distinction in surgery. [6]Distinction in hygiene and preventive medicine.

UNIVERSITY OF WALES

WELSH NATIONAL SCHOOL OF MEDICINE

On the recommendation of the Council of the Welsh National School of Medicine the trustees of the Mansel Talbot Endowment have appointed Fred Grundy, M.D., D.P.H., D.P.M., to the Mansel Talbot Chair of Preventive Medicine in the Welsh National School of Medicine. Dr. Grundy has been medical officer of health to the Borough of Luton since 1937.

UNIVERSITY OF LIVERPOOL

The following candidates have been approved at the examinations indicated:

M.D.—C. V. Donnelly, J. G. P. Jones, Edith M. McConnell, P. L. Robinson, W. Sircus, A. H. Williams.

MASTER OF RADIOLOGY.—*Part I:* J. Ainsworth, Constance M. Davis. *Part II:* J. F. Doherty, W. J. L. Francis, B. B. Harrison, W. Kulke, J. P. MacLaughlin, A. S. Whitehead, F. B. Wright.

M.B., Ch.B.—[1] [2]Brenda M. Heathcote, [1] [2]W. B. Jennett, [1] [2]J. C. Richardson. *Part III (Old Regulations):* Margaret Addison, D. U. Bloor, Rosemary J. Bolam, R. Bradshaw, K. W. Brewster, P. Burrows, Anthea M. Bushby, Jean O. R. Carmichael, Mary I. Charlesworth, Jean M. Collins, Beryl C. Davies, Catherine M. Durkin, W. Ellis, O. L. Evans, Shelagh J. Fair, G. H. Fletcher, R. I. Green, I. W. Griffith, Margaret E. Hall, D. B. Hargreaves, J. F. Hobson, W. M. Hunter, P. Hurst, [2]Margaret L. Johnson, J. L. Jones, R. E. Jones, R. H. Jones, Margaret Knox, Margaret A. Lees, Halina Z. Lewinska, D. H. Lloyd, Marcia McArthur, Audrey M. L. Owens, Ethel Owens, W. R. Parry, S. Plumpton, Sheila M. Pope, Ursula C. Pope, J. N. Rimmer, R. K. Rimmer, Myfanwy R. H. Roberts, E. Rogansky, H. U. Shaw, Pauline R. Sullivan, J. C. Taylor, R. P. Watkins, D. L. Williams, J. A. Williams. *Passed in Separate Subjects:* D. J. Adams, Dorothy M. Broadhurst, and J. S. Endbinder (Surgery, Obstetrics and Gynaecology); Lily I. Chesham and C. Griffith (Medicine, Obstetrics and Gynaecology); A. J. Cunningham, Betsy E. Davies, E. J. J. Davies, and B. J. Maxwell (Medicine, Surgery).

DIPLOMA IN PUBLIC HEALTH.—D. J. Cusiter, Elinor M. Evans, S. Ghousuddin, K. G. Koshi, D. A. W. Nugent, G. P. Reed, H. D. Sutherland, W. F. Wille.

[1]With second-class honours. [2]With distinction in obstetrics and gynaecology.

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

At a special meeting of the Council held at the College House on July 5 with the President, Sir William Gilliatt, in the chair, the Honorary Fellowship of the College was conferred on Sir Henry Dale, O.M., F.R.S., President of the Royal Society of Medicine, and on Professor J. E. Heyman, of Stockholm.

The following were admitted to the Fellowship of the College: H. L. Sheehan, D. F. Standing.

The following were admitted to the Membership of the College: G. C. Brentnall, J. H. Maloney, C. Mukherjee, E. I. Ostry, H. Renton, D. B. Stewart, E. G. Zacks.

The President formally presented the prize for an essay on "The Physiology of the Third Stage of Labour" to Dr. Jean R. C. Burton-Brown; the prize was shared equally with Dr. David Shaw, whose essay was on "The Management of the Third Stage of Labour."

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended July 2. [No. 26

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1949 (a)	(b)	(c)	(d)	(e)	1948 (Corresponding Week) (a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	23	3	15	3		42	5	15	3	—
Deaths		—	1				1	—		
Diphtheria	80	14	37	—	13	141	16	40	11	7
Deaths	—	—	—	1	—	2	1	—	—	—
Dysentery	49	3	61	—		69	9	39	—	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	—	—	—	—		1	—	—	—	—
Deaths		—					—			
Erysipelas			14	9	2			33	6	1
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				55					12	
Deaths	22	3	4	2	—	27	1	11	2	1
Measles*	6,984	714	180	276	137	8,987	680	120	132	65
Deaths†			—	1	—			—	—	1
Ophthalmia neonatorum	50	—	14	1	1	57	1	5	—	—
Deaths										
Paratyphoid fever	13	—	—	—		6	—	1(B)	—	—
Deaths	—	—	—	—	—	—	—	—	—	—
Pneumonia, influenzal	376	19	5	1	8	359	9	4	5	—
Deaths (from influenza)‡	6	—	2	—	—	6	1	—	—	—
Pneumonia, primary			169	38				134	25	
Deaths	135	15		11	3	134	22		4	8
Polio-encephalitis, acute	1	—				2	—			
Deaths										
Poliomyelitis, acute	80	6	2	2	—	36	3	1	2	—
Deaths§	4	2				3	—			
Puerperal fever		—	10				2	9		—
Deaths										
Puerperal pyrexia‖	101	11	13	—	—	120	13	6	—	1
Deaths		—					—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,031	96	146	59	29	1,532	87	353	54	23
Deaths†			—	—	—			—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—					—	—
Typhoid fever	4	2	—	2	1	6	—	—	3	—
Deaths	—	—	—	—		—	—	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—					—	—
Whooping-cough*	2,260	110	52	89	74	2,907	223	18	69	16
Deaths	6	—	1	—	1	6	1	1	3	—
Deaths (0–1 year)	251	37	32	19	11	243	31	41	29	10
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	4,192	707	555	168	106	3,867	607	522	182	118
Annual death rate (per 1,000 persons living)			11·1	10·4				10·5	11·4	
Live births	7,618	1212	891	446	257	8,006	1275	971	485	253
Annual rate per 1,000 persons living			17·9	27·6				19·6	30·3	
Stillbirths	181	18	22			194	17	44		
Rate per 1,000 total births (including stillborn)			24					43		

* Measles and whooping-cough are not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales, Eire, and Northern Ireland.

EPIDEMIOLOGICAL NOTES

Food Poisoning in Yorkshire

On July 2, 332 cases of food poisoning occurred among school-children at Settle, Yorkshire. *Salmonella typhi-murium* was isolated. The symptoms were fever, headache, prostration, bellyache, vomiting, and diarrhoea. In the mildest cases the symptoms were constitutional rather than related to the abdomen, and were attributed at first to the heat of the sun or in one case to teething. Most cases were mild to moderate, but, in the more severe, frequent stools were passed containing blood and mucus. There were no deaths. On June 30 Farm A, which supplied school milk, was short of the required quantity and bought some from Farm B. A cowman at Farm B had been ill with diarrhoea and vomiting during the week but had carried on with his work: it was hay-making time and everyone was very busy. The milk from Farm B was stored overnight in a refrigerator and distributed with milk from Farm A to schools in Settle Rural District on July 1. There were no cases in Skipton Rural District, which received unmixed milk from Farm A, and no cases on Farm A's milk round. There were three mild cases on Farm B's milk round and two moderately severe ones on the farm itself. *Salm. typhi-murium* was isolated from a case on Farm B, from a sample of the school milk supplied on July 1, from four out of six empty but unwashed milk bottles, and from the stools of selected cases among the school-children.

Poliomyelitis

An increase in the number of notifications of poliomyelitis has been reported for four consecutive weeks. The trend of this disease is one of increasing incidence until the autumn, and it seems likely that a large outbreak, such as was experienced in 1947, must be expected. The number of notifications for the 24th to 26th weeks were 31, 44, and 56 in 1947 compared with 35, 48, and 80 of this year.

During the week the largest returns were Cornwall, Camborne–Redruth U.D. 9; Yorkshire West Riding 9; Middlesex 8; London 6 (Lewisham 3); Lancashire 5; Devonshire 4; Oxfordshire 4; Essex 3; Hertfordshire, Hatfield R.D. 3; Gloucestershire 3. In Cornwall the disease has chiefly attacked young children, and three-quarters of the first 12 notifications were of children under 5.

Notifications in the week ended July 9 were poliomyelitis 73 (80), polio-encephalitis 4 (1). (Figures for the previous week are in parentheses.) Multiple cases (poliomyelitis and polio-encephalitis together) were reported from the following counties: London 6 (6), Cheshire 2 (2), Cornwall 5 (9), Devon 11 (4), Gloucestershire 3 (3), Hertfordshire 4 (3), Lancashire 5 (5), Middlesex 5 (9), Nottinghamshire 3 (0), Southampton 2 (1), Warwickshire 3 (1), Yorkshire West Riding 10 (9), Glamorgan 3 (1).

The general picture of incidence is very different from what it was at the corresponding period in 1947. In 1947 the early rise in incidence was chiefly in the more densely populated parts of central England, with additional foci in north-west Lancashire (Barrow) and in Tyneside. This year, although there has been some increase of incidence in the central parts, the chief increases have been in the west and south. Notifications in the West Riding of Yorkshire have risen to 10 (9), but otherwise the north has remained remarkably free from the disease. Another difference between 1949 and 1947 has been high attack rates in relatively small and isolated communities.

Discussion of Table

In *England and Wales* there was an increase in the number of notifications of measles 121, whooping-cough 79, acute pneumonia 42, and acute poliomyelitis 32; decreases were recorded for scarlet fever 106, paratyphoid fever 18, and dysentery 17.

The largest increases in the notifications of measles were Warwickshire 103 and London 101, and the largest decrease was Kent 193. The largest variations in the local returns of whooping-cough were increases in Lancashire 56 and Warwickshire 25 and a decrease in Suffolk 25. The only considerable rise in the number of notifications of scarlet fever was 20 in London, and the largest falls were Lancashire 34, Yorkshire West Riding 33, and Warwickshire 28. There was no great change in the local returns of diphtheria, but the disease was slightly more widespread, and four more counties reported a case than in the preceding week.

A further 9 cases of paratyphoid fever were notified in Durham; the number of notifications in the three preceding weeks were 26, 28, and 15. The only important centre of dysentery was Lancashire 22 (Liverpool C.B. 20).

In *Scotland* decreases were recorded in the number of notifications of measles 121 and scarlet fever 29, while increases were reported for dysentery 18 and diphtheria 13. The rise in incidence of diphtheria was spread throughout the country. The chief centres of dysentery were Glasgow 32, Edinburgh 10, and Midlothian county 5.

In *Eire* increases were reported in the number of notifications of measles 76 and diarrhoea and enteritis 16. The increased incidence of measles was general throughout the country except in Dublin C.B. The largest returns were Dublin C.B. 136, Kerry, Listowel R.D. 47, Cork C.B. 29, and Tipperary, Tipperary U.D. 21. An increase of 10 in the notifications of diarrhoea and enteritis occurred in Dublin C.B.

In *Northern Ireland* a decrease occurred in the number of notifications of measles 16 and scarlet fever 28, while an increase of 8 was recorded for diphtheria.

Week Ending July 9

The notifications of infectious diseases in England and Wales during the week included: scarlet fever 946, whooping-cough 2,183, diphtheria 103, measles 5,571, acute pneumonia 346, cerebrospinal fever 39, acute poliomyelitis 73, dysentery 59, paratyphoid 32, and typhoid 5. Of the 477 cases of food poisoning 332 were notified in Yorkshire West Riding, Settle R.D. (see above).

Medical News

Leverhulme Research Fellowships

The advisory committee has recommended and the trustees of the Leverhulme Research Fellowships have approved the following awards for research tenable for periods up to two years: *Fellowship,* Dr. H. M. Woodman, chemotherapeutic treatment of filariasis in Southern Sudan. *Research Grants,* Dr. G. W. Harris, neural control of the pituitary gland; Dr. D. A. McDonald, the role of the hypothalamus and brain-stem in the regulation of the autonomic nervous system. Application forms and further information may be obtained from the secretary, Leverhulme Research Fellowships, 7, Bedford Row, London, W.C.1.

Hire Purchase Facilities

The Medical Sickness Finance Corporation, whose chairman is Sir Cecil Wakeley, has been incorporated as a private limited company to give hire purchase facilities to medical and dental practitioners wishing to buy cars and equipment. The corporation is a subsidiary of the Medical Sickness Annuity and Life Assurance Society, Ltd. Its address is 7, Cavendish Square, London, W.1.

Society of Medical Officers of Health

Dr. H. C. Maurice Williams, O.B.E., medical officer of health for Southampton, has been elected president of the Society of Medical Officers of Health for the year 1949–50.

Gift of Medical History Journal

Mrs. Josiah C. Trent, of Durham, North Carolina, U.S.A., has instituted an annual gift of 250 one-year subscriptions for the *Journal of the History of Medicine and Allied Sciences.* They will be allocated to all medical schools in the U.S.A. and Canada. Recipients are to be selected from among those in the graduating class who have shown an interest in the cultural aspects of medicine. The late Dr. Josiah C. Trent was a founder of the journal and a member of its board of editors. Recently the journal came under the official sponsorship of Yale University School of Medicine.

International Congress of Otolaryngology

The congress was opened by the Duchess of Kent on July 18 at the Great Hall, King's College, London. Thirty-nine countries were represented.

Tribute to Sir Gordon Gordon-Taylor

The April issue of the *British Journal of Surgery* is dedicated to Sir Gordon Gordon-Taylor, K.B.E., C.B., F.R.C.S., consulting surgeon to the Middlesex Hospital, and includes an appreciation of his work in the field of surgery since he entered the Middlesex Hospital as a student in 1898.

Actonian Prize

The managers of the Royal Institution have awarded the Actonian Prize of one hundred guineas for the year 1949 to Sir Alexander Fleming, F.R.S., for his paper published in 1945 entitled "Penicillin—Its Discovery, Development, and Uses in the Field of Medicine and Surgery." The prize has been awarded every seventh year since 1838 under a trust established in that year by the late Mrs. Hannah Acton.

Lecturing in Germany

Dr. G. M. Findlay, editor of *Abstracts of World Medicine*, is lecturing in Germany at the invitation of the Foreign Office on recent advances in knowledge of virus diseases.

Council for Welfare of Handicapped Persons

Lord Rushcliffe has resigned, owing to ill-health, the chairmanship of the Ministry of Health Advisory Council for the Welfare of Handicapped Persons. The Minister has appointed Mr. Edward Evans, M.P., formerly vice-chairman, in his place. The Minister has also appointed Alderman Mrs. L'Estrange Malone, chairman of the Welfare Committee of the London County Council, to be a member of the Advisory Council in place of Councillor Douglas Prichard, who has resigned.

Laboratory Examinations

The Ministry of Health states that laboratory examinations of pathological material relating to outbreaks or suspected outbreaks of infectious disease, other than those occurring in hospitals in which a pathologist is working, should be referred to the Public Health Laboratory Service where possible and not be undertaken in hospital laboratories. If the Public Health Laboratory Service is not available within a reasonable distance, these examinations should be undertaken by hospital laboratories and charged to hospital funds. Any information of epidemiological interest should be reported to the Public Health Laboratory Service and to the medical officer of health concerned. Examinations of specimens taken from material not of human origin should be undertaken by hospital laboratories only by arrangement with the Public Health Laboratory Service, the latter meeting the cost. Unless the hospital wishes, this arrangement need not apply to samples of water, milk, food, etc., supplied to the hospital for its own use. The Public Health Laboratory Service will not be responsible for the payment of fees to a pathologist in private practice except where he has been called in by arrangement with that service. The term "infectious diseases" excludes venereal diseases and tuberculosis in institutional cases. Examinations in these cases are normally a hospital responsibility and are undertaken by the Public Health Laboratory Service only by special arrangement.

COMING EVENTS

Veterinary Congress

The 14th International Veterinary Congress will be held on Aug. 8–13 at the Central Hall and Church House, Westminster. Subjects to be discussed include the control of bovine tuberculosis, *Brucella abortus* infection, and meat inspection. Information may be obtained from the National Veterinary Medical Association, 36, Gordon Square, London, W.C.1.

Air Medicine

The 20th Annual Convention of the Aero Medical Association will be held at the Hotel Statler, New York City, on Aug. 29–Sept. 1.

South African Medical Congress

The Medical Association of South Africa will hold its 37th medical congress and 16th annual scientific meeting at the University of Capetown, Rondebosch, on Sept. 19–24. Particulars may be obtained from the honorary organizing secretary, 37th S.A. Medical Congress, Medical House, 35, Wale Street, Capetown.

International Society of Surgery

The thirteenth congress of the International Society of Surgery will be held at New Orleans, Louisiana, U.S.A., from Oct. 10 to 15 under the presidency of Professor G. Grey Turner (chairman of the British Executive Committee), when the following subjects will be discussed: "The Surgery and Pathology of the Pancreas, especially in Relation to its Endocrine Function," to be opened by Professor Ian Aird (London); "The Surgery of the Parathyroid Glands," to be opened by Dr. Paolucci (Rome); "The Surgery of the Suprarenal Glands," to be opened by M. R. Fontaine (Strasbourg); "The Pathology and Surgery of the Pituitary Gland," to be opened by M. Paul Martin (Brussels); "The Treatment of Post-operative Thrombosis and its Sequelae," to be opened by Dr. A. Ochsner and Dr. de Bakey (New Orleans); "The Causes of Recurrence after Operations on the Biliary Tract," to be opened by M. R. Demel (Vienna). Members who wish to take part in discussions are reminded that their names should be sent without delay to Dr. Dejardin, Secretary-General, 141, Rue Belliard, Brussels, Belgium. Members are individually responsible for making their own travel arrangements, whether by the route outlined in the official programme or otherwise.

SOCIETIES AND LECTURES

Tuesday

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., July 26, 11 a.m., "*Sociological Problems in Relation to V.D.*," by Dr. R. Lees.

Wednesday

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., July 27, 11 a.m., "*Psychological and Medico-legal Aspects of V.D.*," by Dr. W. N. Mascall.

Thursday

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., July 28, 11 a.m., "*The Problem of Leucorrhoea*," by Dr. W. N. Mascall.

Friday

BIOCHEMICAL SOCIETY.—At Biochemistry Department, University College, Dundee, July 29, 11 a.m., 278th meeting. Papers will be read.

Saturday

MEDICAL SOCIETY FOR THE STUDY OF VENEREAL DISEASES.—At Leicester, July 30. Presidential address.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Essex-Lopresti.—On July 11, 1949, at Birmingham, to Muriel (née Harrop), M.B., B.S., D.R.C.O.G., wife of Mr. P. Essex-Lopresti, F.R.C.S., a sister for Timothy—Catherine.

Harper.—On July 5, 1949, at Swindon, to Dr. Marita Harper (née Shattock), wife of Dr. Eric Harper, a daughter.

McDonagh.—On July 10, 1949, to Dr. and Mrs. McDonagh, Stanley, Perth, a daughter.

MARRIAGES

Brennan—Reed.—On June 18, 1949, at St. Marks, North Audley Street, London, W., Squadron Leader Terence N. N. Brennan, M.B., B.Ch., to Mrs. Betty Reed, Cambridge, widow of the late Lieutenant E. J. Reed.

DEATHS

Brown.—On July 13, 1949, at Grosvenor Villa, Gullane, East Lothian, Robert Archibald Brown, M.B., Ch.B., Surgeon Captain R.N., retired.

Campbell.—On June 19, 1949, at Phoenix House, Sutton Oak, St. Helens, Lancs, Donald Campbell, M.R.C.P., aged 66.

Carre-Smith.—On July 14, 1949, in London, Herbert Lovell Carre-Smith, M.R.C.S., L.R.C.P., aged 80.

Collier.—On July 11, 1949, at Lancing, Sussex, William Arthur Collier, L.R.C.P., L.R.C.S.Ed., aged 82.

Cooke.—On July 13, 1949, at 48, Kingston House, London, S.W., Sir James Douglas Cooke, F.R.C.S., aged 70.

Fisher.—On July 13, 1949, at Lynton, Cheltenham Road, Gloucester, Theodore Fisher, M.D., F.R.C.P., aged 86.

Gerrie.—On July 4, 1949 at Harrogate, John Gerrie, M.B., Ch.B., F.R.C.S.Ed., of 2, Queen's Gardens, Aberdeen.

Gourlay.—On July 15, 1949, at Rathmore, Elgin, Frederick Gourlay, M.D., in his 82nd year.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Vitamin C in Fresh Fruit

Q.—*Is there any evidence to support the view that the vitamin-C content of fresh fruit is mainly to be found just under the peel or skin or actually in it? Is it a fact that bananas and fresh strawberries contain negligible amounts of vitamin C?*

A.—There is experimental evidence that the vitamin C of apples is found mainly in the tissues underneath the skin. The vitamin-C content diminishes fairly rapidly from the skin inwards. The amount in the cuticle is not great, but much would be removed in peeling in the ordinary way. There is little experimental evidence about the vitamin-C content of different parts of the pear, which is poor in this substance. The question would not apply to fruits like the orange, or to soft fruits which are wholly edible. The vitamin-C content of apples varies widely from about 25 mg. per 100 g. for the Bramley Seedling to about 8 mg. or less for the Worcester Pearmain. The banana has about the same amount of vitamin C as apples of the latter type. Of native fruits the strawberry is one of the richest in vitamin C, with about 60 mg. per 100 g.—that is, second only to the blackcurrant, with about 200 mg. It is, on the average, superior to the orange, with about 50 mg.

Epidermophytosis

Q.—*What is the risk of contagion from epidermophytosis between the toes? The problem arises especially in connexion with children sharing bedrooms during summer holidays. Is the risk lessened as time passes? What is the treatment, and is any form of prophylaxis possible?*

A.—It should be mentioned that the majority of patients referred to dermatologists as suffering from epidermophytosis prove to have pompholyx of constitutional origin. Confirmation of diagnosis by microscopical examination and culture is desirable. Risk of contagion is great, especially through contact in bed. It is much reduced by parasiticidal measures, the wearing of protective socks in bed and of sandals about the house and bathroom, and the use of toilet powders containing 2% salicylic and boric acids in talcum between the toes by day. Reference may be made to questions in the *Journal* of June 5, 1948, p. 1120; Jan. 22, 1949, p. 164; and Feb. 19, 1949, p. 331, concerning other prophylactic measures. Modern treatment with fatty acids and their esters, such as undecylenic, propionic, and caprylic acids, is very effective. There are many suitable preparations on the market.

Complications of Treatment of Varicose Veins

Q.—*Could you give me any statistics relating to the incidence of pulmonary embolism and other potentially fatal complications following ligation and injection treatment of varicose veins? Is it customary to keep the patient in bed for any length of time after the operation, or is the modern method to allow active exercise immediately? Also are anticoagulants needed post-operatively?*

A.—Statistics on this point are not readily obtainable, but nevertheless deep femoral thrombosis and fatal pulmonary embolus do occur and are reported at coroners' inquests. Further causes of death are anaphylaxis and suppression of urine, which may be due to the injected solution. Violent haemorrhage from the slipping of a ligature may also be directly fatal or predispose to later embolism. Ambulation immediately after operation is imperative if deep thrombosis and embolism are to be avoided. Operation and injection should not be undertaken in those whose family and personal history reveals several repeated thromboses so easily provoked as to justify the diagnosis of thrombophilia. Intervention in these patients may cause death or disablement from venous thrombosis, or may be followed by arterial involvement with loss of a limb.

Anticoagulants are essential if deep thrombosis or embolism has occurred, starting with 45,000 units of heparin and 300 mg. of dicoumarol for two days and then continuing with amounts of dicoumarol adequate to increase the prothrombin time by 120%. When the clotting is under control, ambulation can be resumed.

Bram's Test for Hyperthyroidism

Q.—*What is Bram's test for hyperthyroidism, and how is it applied?*

A.—Bram's test for hyperthyroidism depends on his observation (*Med. Rec., N.Y.*, 1920, **98**, 887) that 95% of thyrotoxic patients can tolerate large doses of quinine without evidence of cinchonism. In his book *Exophthalmic Goitre and its Medical Treatment* (London, Henry Kimpton, 1936) he suggests that the patient should be given 12 capsules, each containing 10 gr. (0.65 g.) of quinine hydrobromide or sulphate. The patient is instructed to take one of these capsules three times daily, and the non-appearance of symptoms of cinchonism within four days is considered as a positive reaction. Bram obtained a negative reaction in only 4.25% of thyrotoxic patients, and he believed that tolerance of quinine and basal metabolism run parallel.

Muscae Volitantes

Q.—*Is there any general or local treatment for muscae volitantes?*

A.—Muscae volitantes are generally of a septic, haemorrhagic, or degenerative origin. In the first two instances successful treatment of the cause will prevent further increase in the muscae. The elimination of muscae is a difficult matter. Potassium iodide by mouth is sometimes useful, and the muscae are much less obvious if glasses properly correcting the focus are worn. Subconjunctival injections of hypertonic saline, etc., seem to have no effect. In traumatic cases with a haemorrhagic origin aspiration of a moderate quantity of vitreous sometimes helps (50% of cases in the writer's experience). For the type due to degeneration of the vitreous no treatment is effective.

Intestinal Haemorrhages

Q.—*Within 12 months an unmarried female aged 24 has had three severe intestinal haemorrhages, which occurred without warning. There is no history of dyspepsia, nor has she any abdominal symptoms. No abnormality has been found on x ray or proctoscopy, or at laparotomy. She has suffered from a minor degree of spondylitis for the past 10 years. Can you suggest a diagnosis and treatment?*

A.—The most likely cause of severe intestinal haemorrhage in a woman of 24 is ulceration associated with a Meckel's diverticulum, but this presumably has been excluded by laparotomy. Other possibilities include hiatus hernia of the stomach—in these cases haematemesis would be more likely than melaena—and simple tumour of the small intestine; lipoma, fibroma, and leiomyoma can all cause recurrent haemorrhage and, if small, might be missed at laparotomy. Telangiectasia of the gut, if part of the familial haemorrhagic telangiectasia (Rendu-Osler-Weber disease), is likely to be associated with epistaxis.

Further investigation of this case should include a barium meal for small-intestinal pattern and to exclude hiatus hernia; gastroscopy; examination of stools for occult blood to see whether the bleeding persists; and, should massive haemorrhage occur again, a laparotomy at the time of bleeding so that the level of haemorrhage in the gut can be ascertained. The following references may be useful: Eads, J. T., 1946, *J. Amer. med. Ass.*, **131**, 891; Kennedy, H. P., 1947, *New Engl. J. Med.*, **237**, 180; Allison, T. D., and Babcock, J. R., 1948, *Ann. Surg.*, **127**, 754; Baker, H. L., and Halley, H., 1946, *Ann. Surg.*, **123**, 1067.

Valsalva Experiment

Q.—*Can you give the physiological explanation of the after-dinner trick whereby the victim is rendered temporarily unconscious by strong compression of his chest, held fully expanded in deep inspiration, by the interlocked encircling arms of the operator? I have seen this performed only once, when the victim became unconscious for a few seconds after compression for about half a minute. He gave a slight convulsive jerk before regaining consciousness, which was immediate and full. The pulse was slow, full, and regular, and there was no after-effect or ill feeling of any sort. I have found no record of tragedy, but I feel that this procedure must be potentially dangerous.*

A.—It is possible to render a victim unconscious by strong compression of the chest aided by his natural reaction, which consists of forced expiration against a closed glottis. This is the Valsalva experiment, and if carried out sufficiently vigorously it halts the circulation, the intrathoracic pressure being so greatly raised as to prevent cardiac filling. In such circumstances loss of consciousness must be associated with absence of the pulse. The procedure is unlikely to be dangerous in healthy subjects, for the circulation is fully restored immediately the individual is released.

Delirium Tremens

Q.—*What is the best treatment for delirium tremens?*

A.—It is perhaps best to tide the patient over with small doses of alcohol and intramuscular injections of paraldehyde in doses of 2 dr. (8 g.) repeated at four-hourly intervals if necessary. Any form of adequate sedation will relieve delirium.

Persistent Abdominal Sinus

Q.—*About a year ago a classical caesarean section was carried out in a woman of 22 with contracted pelvis due to spastic hemiplegia. The uterus was emptied and sutured with silk mattress sutures. The puerperium was uneventful except for a stitch abscess in the abdomen, which resolved after treatment by fomentations. The uterus involuted satisfactorily, though there is slight menorrhagia. After three months another abscess developed in one of the abdominal sutures and a piece of catgut was extruded; this was followed by a sinus which has persisted, and through which bleeding has occurred during the last three menstrual periods. Recently one of the uterine sutures was extruded through the sinus, which still persists and remains troublesome. What is the prognosis, and what further steps do you recommend?*

A.—This patient would appear to have either a uterine fistula or an endometrioma of the abdominal wall, or both. A spontaneous cure is now unlikely, and there is little to be said for continuing conservative measures. Any operation, however, should be conservative in nature and should aim at complete excision of the scar and sinus and the restoration of normal uterine function. Preliminary studies by means of radio-opaque media injected along the sinus track, and also into the uterine cavity through the cervix, would probably be helpful. The operation is likely to be difficult and should be carried out by an experienced gynaecologist.

NOTES AND COMMENTS

Dicoumarol and Prothrombin Level.—Dr. G. A. MATTHEWS (Hemel Hempstead, Herts) writes: I was surprised to see in "Any Questions?" (July 2, p. 38) an answer mentioning the use of Russell viper venom for measuring the prothrombin time of patients on dicoumarol therapy. It has been conclusively shown[1] that Russell viper venom, when used as a thromboplastin, does not measure the prothrombin content of plasma, and that the coagulation times obtained with this thromboplastin bear no recognizable relationship to the availability of prothrombin in a patient under treatment with dicoumarol. It follows from this that to attempt to control dicoumarol therapy by means of Russell viper venom as a thromboplastin can be positively dangerous, as several workers have discovered,[2 3] though the blame is often wrongly put on the use of dicoumarol. Dicoumarol therapy is a safe procedure when brain thromboplastin is used, as in Quick's one-stage technique of measuring prothrombin times, as has been shown in my laboratory during the past two years. I trust, Sir, that you will see that steps are taken to correct the dangerous advocacy of a discredited method of controlling dicoumarol therapy.

Dr. A. F. MOHUN (London, N.19) writes: There is now sufficient evidence available to justify the statement that the use of viper venom as a source of thromboplastin is fraught with danger to the patient, in that this reagent may on occasion fail to disclose that the prothrombin time is in fact approaching, or has already reached, a dangerous prolongation. Alternative reagents have hitherto consisted of saline extracts of acetone-dried human or rabbit tissue (usually brain). These are free from what has become known as the "viper venom effect." An even simpler form of extract has been devised by Dr. M. Toohey, of New End Hospital, Hampstead (personal communication). I understand that an account of this is in the press. Finally, I should like to express my personal opinion that your reply to the question would have performed a real service to clinicians and pathologists if it had confined itself solely to the statement that the control of dicoumarol therapy is achieved by aiming at keeping the patient's prothrombin time within the range of one and a half to two and a half times the normal control time, using the same reagents. It is still not sufficiently appreciated that "prothrombin indices" and "prothrombin percentages" are calculations whose only basis is the measurement of time. No advantage is gained by elaboration of the basic data. The calculating of "prothrombin percentages" implies that prothrombin is a chemical individual, and that saline dilutions of normal plasma truly imitate plasmas from patients receiving dicoumarol: neither of these implications can be substantiated—they are therefore not only unnecessary but possibly misleading. Although the chemistry of the prothrombin time reaction is obscure, the principles of the control of dicoumarol treatment are clear and well substantiated: Use a potent thromboplastin other than viper venom; measure the prothrombin time of the patient's plasma and that of a normal control plasma (both within three to four hours of removal of the blood); regulate the dosage of dicoumarol with the aim that the time ratio shall lie between one and a half and two and a half. The method of collection of the blood and the interpretation of the results call for personal collaboration between pathologist and clinician.

REFERENCES

1 Biggs, R., and Macfarlane, R. G. (1949). *J. clin. Path.*, **2**, 33.
2 Lempert, H. (1948). *British Medical Journal*, **1**, 125.
3 James, G. A. (1948). Ibid., **1**, 475.

Transient Giddiness.—Dr. DAVID COWAN (Johannesburg) writes: In "Any Questions?" (May 28, p. 968) a question was asked on the causes and treatment of momentary transient vertigo. A possible cause not mentioned in your answer, and one I have met with on several occasions, is that of cerumen in the ear, deep enough presumably to cause pressure on the tympanum. Syringing gives complete relief.

Corrections

The report of the meeting of the Section of Ophthalmology held on June 30, which appeared in our issue of July 9 (p. 92), incorrectly summarized Dr. Alice Carleton's views. The last sentence reporting her observations does not represent her opinion.

In the account of the combined meeting of the Sections of Obstetrics and Gynaecology and Dermatology held on June 30, which appeared in our issue of July 9 (p. 88), Dr. Elizabeth Hunt is incorrectly reported to have said that in her experience psychogenic causes of pruritus vulvae were rare. In fact she said that pruritus vulvae due solely to psychogenic causes was in her experience rare.

In the report (July 16, p. 165) of Dr. W. Pointon Dick's remarks during the discussion on tuberculosis and occupation at the meeting of the Section of Occupational Health it was stated: "In return visits it was found that the rate of new cases per 100 examined was reduced by a third." This should have read "reduced to a third."

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